ALICE MARGARIDA CORREIA DOS SANTOS DA SILVA

EFFECT OF LOWER LUMBAR HERNIATION ON ADJACENT LEVELS

Master's Dissertation Report in Biomedical Engineering

Supervisor

Professor Doutor André Castro

Co-supervisor

Dr. Nuno Cristino

December 2025

ALICE
MARGARIDA
CORREIA DOS
SANTOS DA SILVA

EFFECT OF LOWER LUMBAR HERNIATION ON ADJACENT LEVELS

EXAMINATION BOARD

President: (Doutor, Célio Pina, Instituto Politécnico de Setúbal)

Supervisor: (Doutor, André Castro, Instituto Politécnico de Setúbal)

Supervisor: (Dr., Nuno Cristino, Hospital da Luz de Setúbal)

Examiner: (Doutor, Ivo Roupa, Instituto Superior Técnico)

December 2025

ACKNOWLEDGEMENTS

First and foremost, I would like to express my sincere gratitude to the Polytechnic Institute of Setúbal (IPS) for these five years, and the opportunities provided throughout my academic journey. To all the professors, staff, and colleagues who contributed, directly or indirectly, to my academic and personal growth. The learning environment, the encouragement, and the dedication of everyone involved were essential for the completion of this work and for the enrichment of my overall academic experience.

A special thank you to my supervisors, Prof. André Castro and Dr. Nuno Cristino, for their continuous guidance, precious knowledge, perceptive ideas, and above all, their patience and support, without which this work would not have been possible.

Also, a huge thank you to my colleague Inês Mendes, who guided me, helped me, and above all, supported me when I needed it the most.

My sincere appreciation also goes to Hospital da Luz de Setúbal for giving me the opportunity to carry out this dissertation, and to the staff, Technician Carlos Martinho and Nurses Mafalda Branco and Marisa Oliveira, for their availability, collaboration, and willingness to help.

To my friends, a heartfelt thank you for accompanying me throughout all my school years, for the good and the bad memories we created together, and for your friendship and encouragement, which made this journey so much more meaningful.

Finally, to my family and my boyfriend João, thank you from the bottom of my heart for your endless patience, strength, and unconditional support, and for always believing in me, even in the most challenging moments.

ABSTRACT

Lumbar disc herniation (LDH) is a degenerative condition and a major cause of low back pain and radicular symptoms. Although its pathophysiology is well understood, the impact of LDH on adjacent discs remains insufficiently clarified.

This dissertation investigates how LDH at L4-L5, L5-S1, or both levels affects adjacent discs. Twenty-six patients underwent MRI morphometric assessment and qualitative grading using the Pfirrmann classification.

The results showed that the herniated discs presented the most pronounced structural changes, including marked reductions in posterior disc height and the highest Pfirrmann grades. In contrast, the adjacent discs exhibited only minor and irregular variations in quantitative parameters, with no consistent trend in height or angulation across the sample. Pfirrmann grades of the adjacent discs were frequently higher than more cranial levels, particularly in patients with L4-L5 herniation. Patients with two-level herniation demonstrated a broader pattern of degeneration, although the most severe changes remained centred on the symptomatic discs.

Overall, the results show that LDH causes significant degeneration at the affected segment, while adjacent-level involvement is less pronounced than at the herniated disc, it is consistently detectable through qualitative Pfirrmann grading rather than quantitative morphometric measures.

Keywords: Lumbar spine, lumbar disc herniation, magnetic resonance imaging, adjacent levels.

RESUMO

A hérnia discal lombar (HDL) é uma condição degenerativa e uma das principais causas de lombalgia e sintomas radiculares. Embora a sua fisiopatologia seja bem compreendida, o impacto da HDL nos discos adjacentes permanece insuficientemente esclarecido.

Esta dissertação investiga de que forma a HDL nos níveis L4-L5, L5-S1, ou em ambos, afeta os discos adjacentes. Vinte e seis pacientes foram submetidos a avaliação morfométrica por RM e a classificação qualitativa segundo Pfirrmann.

Os resultados mostraram que os discos herniados apresentaram as alterações estruturais mais pronunciadas, incluindo reduções na altura posterior do disco e os graus de Pfirrmann mais elevados. Contrariamente, os discos adjacentes exibiram apenas pequenas variações quantitativas e irregulares, sem uma tendência consistente ao longo da amostra. Os graus de Pfirrmann dos discos adjacentes foram frequentemente superiores aos dos níveis mais superiores, especialmente em pacientes com hérnia L4-L5. Os pacientes com hérnia em dois níveis apresentaram um padrão degenerativo mais abrangente, embora as alterações mais severas permanecessem centradas nos discos sintomáticos.

De modo geral, os resultados mostram que a HDL provoca degeneração significativa no segmento afetado, e embora o impacto no nível adjacente seja menos pronunciado que no disco herniado, é consistentemente detetável através da classificação qualitativa de Pfirrmann.

Palavras-chave: Coluna lombar, hérnia discal lombar, ressonância magnética, níveis adjacentes.

CONTENTS

LIST OF FIGURES

LIST OF TABLES

ACRONYMS AND ABBREVIATIONS

2D	Two-Dimensional
3D	Three-Dimensional
BMI	Body Mass Index
CAD	Computer-Aided Diagnosis
CNN	Convolutional Neural Network
CT	Computed Tomography
DB	Database
ESI	Epidural Steroid Injection
FEM	Finite Element Method
HLS	Hospital da Luz de Setúbal
IVD	Intervertebral Disc
LBP	Low Back Pain
LDH	Lumbar Disc Herniation
LLA	Lumbar Lordosis Angle
MPR	Multiplanar Reconstruction
MRI	Magnetic Resonance Imaging
NSAID	Non-Steroidal Anti-Inflammatory Drug
PINN	Physics-Informed Neural Network
U-Net	Convolutional Neural Network Architecture for Image Segmentation

CHAPTER 1

INTRODUCTION

Low back pain (LBP) refers to discomfort originating from the vertebral column or surrounding soft tissues. Chronic mechanical LBP is often associated with repetitive trauma or overuse, especially in occupational settings. Most patients experiencing functionally limiting LBP tend to suffer recurrent episodes. Chronic LBP affects up to 23% of the global population, and estimates indicate that 24% to 80% of patients experience recurrence within one year [1].

Lumbar radiculopathy is a syndrome characterized by pain resulting from compression or irritation of the nerve roots at the lumbar region. It is commonly caused by lumbar disc herniation, vertebral degeneration, or narrowing of the intervertebral foramina through which the nerves exit the spinal canal [2].

Lumbar disc herniation (LDH) is a pathological condition characterized by the displacement of the nucleus pulposus through the annulus fibrosus, typically in a posterior or posterolateral direction. This herniation leads to narrowing of the vertebral canal or neural foramina, resulting in mechanical compression of the thecal sac or adjacent nerve roots [3]. The incidence of this condition is approximately 5 to 20 cases per 1,000 adults per year. Most affected individuals are between 30 and 50 years of age, with a higher prevalence in males. The majority of herniations occur in the lower lumbar spine, with 95% of cases located at the L4-L5 and L5-S1 levels. Herniations at higher levels are more frequently observed in individuals over the age of 55 [4]. Several risk factors are known to predispose individuals to this condition, including obesity, smoking, diabetes, connective tissue disorders, and genetic predisposition. In many middle-aged and elderly patients, LDH is associated with spondylosis, contributing to symptomatic nerve compression due to reduced space available for neural structures [5].

Accurate diagnosis of LDH is essential for guiding appropriate clinical management and improving patient outcomes. Magnetic Resonance Imaging (MRI) demonstrates higher inter-observer reliability compared to other imaging modalities and is more effective than Computed Tomography (CT) in differentiating between inflammatory, malignant, or infectious causes of LDH [3].

1.1. Motivation

This research work aims to enhance the understanding and management of LDH, particularly in what concerns to the consequences on the adjacent levels. The main motivation of this study is the need to improve the evaluation and treatment of lumbar spine conditions, particularly LDH, a leading cause of chronic low back pain and neurological impairment that significantly affects quality of life and functional capacity. In addition, recent evidence indicates that long-standing disc pathology may induce biomechanical alterations in adjacent levels even without surgical intervention, potentially predisposing individuals to adjacent segment degeneration. This highlights the importance of investigating how degeneration evolves across lumbar levels and reinforces the relevance of analysing disc and vertebral morphology in patients with LDH.

1.2. Objectives and Research Questions

The main objective of this study is to analyse and correlate quantitative and qualitative MRI-based parameters in patients with lumbar disc herniation (LDH) affecting the last one or two lumbar segments, with particular emphasis on identifying structural alterations in the intervertebral discs adjacent to the herniated level. Additionally, this study aims to characterise morphological patterns of degeneration across the lumbar spine and to assess the influence of demographic factors on disc and vertebral parameters.

Accordingly, this study addresses the following research questions:

1. Do intervertebral discs adjacent to a lower lumbar disc herniation (L4-L5 and/or L5-S1) exhibit measurable structural alterations when compared with non-adjacent lumbar levels?
2. Are qualitative degenerative changes, assessed by the Pfirrmann classification, more sensitive than quantitative morphometric parameters in detecting degeneration at adjacent disc levels?
3. Does the pattern and extent of adjacent-level degeneration differ between patients with single-level and two-level lower LDH?
4. Are manual MRI-based measurements of lumbar spine morphometric parameters reliable and reproducible across repeated assessments and observers?

1.1. Structure of the Dissertation

This dissertation is organised into four main chapters with corresponding sub-chapters, a references section, and 5 appendices. A brief overview of each chapter is presented below:

- Chapter 2: contains the literature review presenting a comprehensive review of the relevant literature regarding the anatomy of the lumbar spine, the pathophysiology of LDH, diagnostic imaging modalities, and biomechanical and morphological considerations. It also discusses previous studies that have

explored the relationship between disc degeneration and adjacent segment alterations.

- Chapter 3: Describes the study design, including the selection of participants, data acquisition, and image analysis procedures. It details the quantitative and qualitative parameters obtained from MRI and explains the measurements made. The criteria for group classification and statistical analysis are also presented.
- Chapter 4: Presents the results obtained from the morphometric analysis and the Pfirrmann classification of disc degeneration. The results are compared across the three study groups and discussed in the context of existing literature. The relationships between morphometric parameters and clinical variables are analysed to interpret the potential impact of LDH on adjacent levels.
- Chapter 5: Summarises the main conclusions drawn from the study and highlights the scientific and clinical implications of the findings. It also outlines the limitations of the work and proposes directions for future research in the field of spinal morphology and degenerative disc disease.

CHAPTER 2

LITERATURE REVIEW

This review is structured into four main sections: Anatomy of the Vertebral Column, Degenerative Spinal Diseases, Lumbar Disc Herniation, and Diagnostic Methods and Analysis. Each section provides a critical analysis of the current knowledge in the field, highlighting relevant findings and identifying gaps for further research.

2.1. Anatomy of the Vertebral Column

The vertebral column (Figure 2.1) is a central anatomical structure characterized by both strength and flexibility. Superiorly, it articulates with the base of the skull, providing crucial support. Inferiorly, it connects bilaterally with the corresponding iliac bones of the pelvis. Structurally, the vertebral column is divided into five distinct regions: cervical, thoracic, lumbar, sacral, and coccygeal. It consists of a total of 33 vertebrae - seven cervical, twelve thoracic, five lumbar, the sacrum (formed by the fusion of five vertebrae), and the coccyx (typically four fused vertebrae).

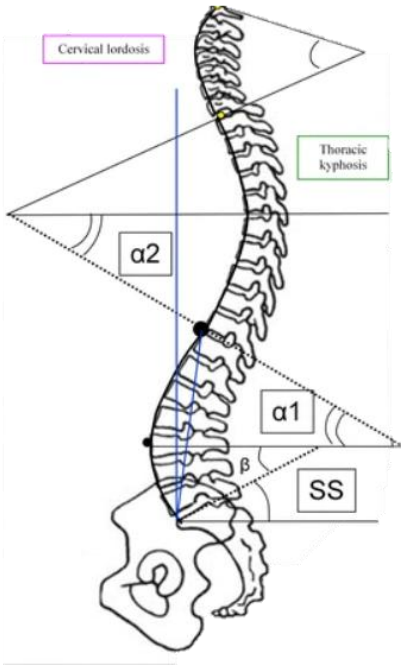

Figure 2.1 - Different types of spine curvature. Adapted from Roussouly et al. 2011 [6].

The vertebral column also features two primary types of curvature: kyphosis, referred to as a primary curvature, and lordosis, known as a secondary curvature. Kyphosis is present in the thoracic region (T1-T12) and the sacral region (S1-S5), where the spine curves posteriorly,

forming an outward convexity. Lordosis curves (L1-L5) anteriorly, creating an inward concavity. Together, these curvatures allow the spine to effectively distribute loads, maintain balance, and provide flexibility throughout movement [6].

2.1.1. Vertebrae

Each vertebra is composed of an anterior vertebral body and a posterior vertebral arch, which together enclose the vertebral foramen. The alignment of successive foramina forms the vertebral canal, which houses and protects the spinal cord and its meninges. The size of the vertebrae gradually increases from the cervical region to the lumbar region, reflecting the progressive demand for weight-bearing capacity, and decreases again in the coccygeal region [7].

The vertebral body has a cylindrical shape and consists of trabecular bone surrounded by a thin cortical layer. The vertebral arch, formed by the pedicles and laminae, gives rise to several osseous projections. The spinous process projects posteriorly, while the transverse processes extend laterally. Additionally, the superior and inferior articular processes, located at the junction between the pedicle, lamina, and transverse process, articulate with adjacent vertebrae, ensuring both mobility and structural stability (Figure 2.2) [7].

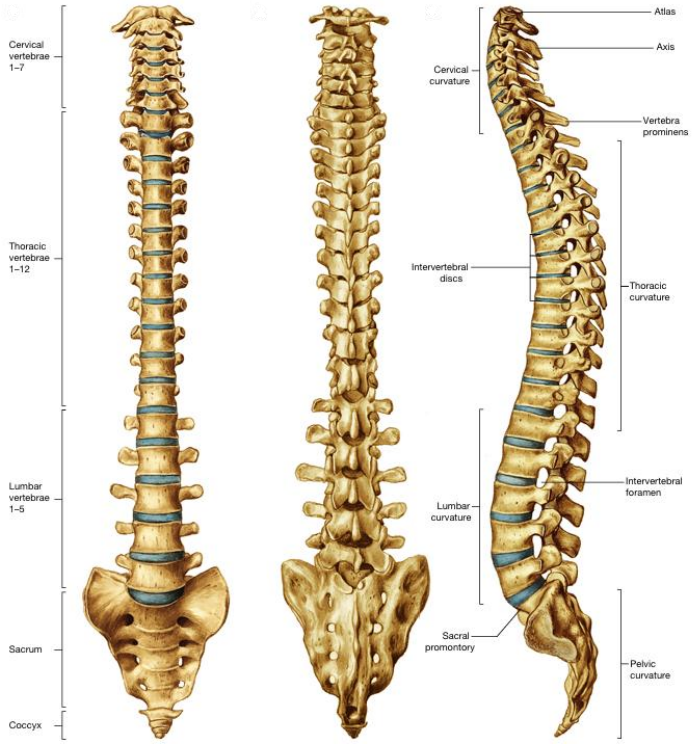

Figure 2.2 - Vertebral column in anterior, posterior and lateral views. Adapted from Mahadevan 2018 [8].

The stacking of vertebrae forms a continuous canal through which the spinal cord passes. Meanwhile, the intervertebral foramina, formed by notches in the pedicles of adjacent vertebrae, serve as passageways for spinal nerves and blood vessels that are vital for the innervation and

vascular supply of the vertebral column. At the junction where the lamina, pedicle, and transverse process meet, the superior articular process projects upward, while the inferior articular process is positioned directly beneath it, allowing precise alignment with adjacent vertebrae [7], [9].

The lumbar region constitutes the lower portion of the vertebral column, extending from the twelfth thoracic vertebra (T12) to the first sacral vertebra (S1). This section is composed of five vertebrae (L1-L5) that are both robust and highly mobile. Their primary function is to dissipate axial forces transmitted through the spine. Owing to their greater size relative to vertebrae in other spinal regions, the lumbar vertebrae are particularly well-suited for bearing and absorbing the loads generated by the head, neck, and upper trunk [10].

In addition to load bearing, the lumbar spine plays a critical role in facilitating trunk mobility. It allows for a wide range of movements, including flexion, extension, rotation, and lateral bending. Viewed in the sagittal plane, the lumbar spine displays a anterior concave curvature known as lumbar lordosis, which can vary in degree among individuals. This curvature promotes optimal weight distribution over the pelvis, contributing to a stable and balanced gait [10].

2.1.2. Intervertebral Discs

Intervertebral discs (IVDs) serve as the primary connective structures between adjacent vertebral bodies, playing a critical role in maintaining both the stability and mobility of the vertebral column. There are a total of 23 IVDs, with the first located between the C2 and C3 vertebrae and the last at the lumbosacral junction. These discs constitute approximately one-third of the total height of the vertebral column and vary in thickness across different spinal regions, being thinner in the upper thoracic region and thicker in the lumbar region. In the lower lumbar segments, the discs acquire a wedge shape, with greater anterior thickness and narrower posterior height, thereby contributing to the natural curvature of lumbar lordosis [8], [9].

Each IVD is composed of two distinct components: the outer annulus fibrosus and the inner nucleus pulposus. The annulus fibrosus consists of concentric layers of collagen fibres that provide high tensile strength and resistance to shearing and torsional forces. In contrast, the nucleus pulposus is a gelatinous, highly hydrated matrix that facilitates the absorption and distribution of compressive loads applied to the spine. With aging, the water content of the nucleus pulposus progressively declines, potentially leading to disc degeneration. This degeneration can result in a reduction in disc height and a subsequent loss of mechanical function [8], [11].

The structural integrity of the IVDs is further supported by two major longitudinal ligaments. The anterior longitudinal ligament extends along the anterior surface of the vertebral bodies and IVDs, while the posterior longitudinal ligament runs along the posterior surface. These ligaments play a crucial role in limiting vertebral motion and protecting the spinal cord [8], [11].

2.1.3. Facet Joints

Facet joints, or zygapophyseal joints, form paired synovial joints between adjacent vertebrae along the entire spine. Each joint is created by the inferior articular process of a superior vertebra and the superior articular process of the vertebra below, allowing region-specific motion. In the lumbar spine, their orientation permits flexion, extension, and lateral bending while restricting rotation, whereas in the thoracic region they allow a wider range of movements, though overall mobility is limited by the rib cage [11].

Each joint is enclosed by a fibrous capsule lined with synovium, and the articular surfaces are covered by hyaline cartilage to ensure smooth movement and contribute to spinal stability. Facet joints receive innervation from the dorsal rami of adjacent spinal nerves, making them a common source of acute and chronic low back pain. With ageing, they may undergo degenerative changes, such as joint space narrowing, cartilage loss, and subchondral hypertrophy, or develop osteoarthritis, all of which can reduce mobility and contribute to pain syndromes [11].

2.2. Degenerative Spinal Diseases

Although various causes can lead to physiological (age-related) and pathological degeneration of bone and joints, repeated minor trauma remains the principal mechanism implicated in spinal degeneration. The concept of "duration trauma" refers to forces that, while insufficient to cause fractures acutely, can still produce bone or disc damage when applied either suddenly or over prolonged periods. Another important aspect in the degenerative process is the functional integrity of spinal curvatures. The presence of four physiological curves in the spine enhances its capacity to absorb trauma more efficiently than a straight structure. When these curvatures are altered, or there is misalignment, asymmetric load distribution may lead to degenerative changes [12].

LBP refers to discomfort originating intrinsically from the vertebral column or surrounding soft tissues. It encompasses a range of conditions including lumbosacral muscle strain, disc herniation, lumbar spondylosis, spondylolisthesis, spondylolysis, vertebral compression fractures, and both acute and chronic traumatic injuries. Chronic mechanical LBP is often associated with repetitive trauma or overuse, especially in occupational settings. Most patients experiencing functionally limiting LBP tend to suffer recurrent episodes. Chronic LBP affects up to 23% of the global population, and estimates indicate that 24% to 80% of patients experience recurrence within one year. Despite significant increases in healthcare spending, particularly in the use of epidural corticosteroid injections (629%), opioid prescriptions (423%), magnetic resonance imaging, and spinal fusion surgeries, only minor improvements were seen in clinical outcomes or disability rates among affected patients [1].

Sciatica is characterized by radiating pain that follows a dermatomal distribution (Figure 2.3), often accompanied by sensory symptoms. Despite its prevalence, the diagnostic value of patient history and physical examination in cases of sciatica remains underexplored. Patients report pain radiating down the leg. No single element of clinical history or physical examination

simultaneously achieves high sensitivity and specificity. The straight leg raise test is the most used physical test, along with the crossed straight leg raise test, although both have limited sensitivity.

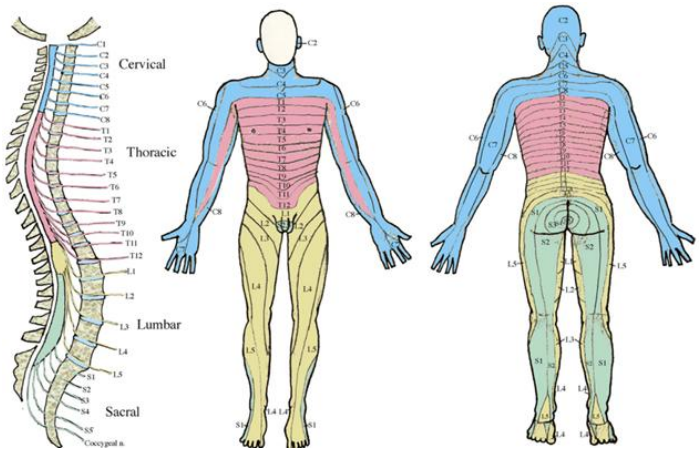

Figure 2.3 - Dermatomal Pattern. Adapted from Zhang et al. 2023 [5].

In general, the diagnosis of sciatica is supported when a patient indicates typical unilateral radiating leg pain, along with one or more positive neurological tests suggesting nerve root tension or deficit. Key signs and symptoms that distinguish sciatica from nonspecific LBP include unilateral leg pain more severe than lumbar pain, radiation of pain to the foot or toes, numbness or paraesthesia in the same distribution, pain triggered by the straight leg raise test, and neurological deficits associated with a single nerve root [13].

Lumbar radiculopathy is a syndrome characterized by pain resulting from compression or irritation of the nerve roots in the lumbar region. It is commonly caused by LDH, vertebral degeneration, or narrowing of the intervertebral foramina through which the nerves exit the spinal canal [2], [3]. Age is a major risk factor, as the condition stems from degenerative processes within the spine. Symptoms typically manifest in middle age, with men more frequently affected in their 40s, and women more commonly between 50 and 60 years of age. Women face an increased risk in certain physically demanding professions, such as the military. However, in the general population, the condition is more prevalent among men [14]. Clinical presentation includes lower back pain radiating to the lower extremities along a dermatomal pattern. Additional symptoms may include numbness, weakness, and loss of reflexes, although their absence does not rule out a diagnosis of lumbar radiculopathy [2].

The Cobb angle is the gold standard for quantifying spinal curvature. It is defined as the angle between lines drawn along the superior endplate of the most tilted vertebra above the curve's apex and the inferior endplate of the most tilted vertebra below it. Although scoliosis is traditionally diagnosed at angles greater than 10°, recent evidence shows that smaller deviations may still be clinically relevant. According to Jin et al., 2022, angles as low as 5° can indicate early asymmetry, particularly in screening or longitudinal follow-up. However, manual Cobb measurements remain vulnerable to inter- and intra-observer variability due to challenges in identifying vertebral landmarks and inconsistencies in image acquisition. These limitations may

lead to over- or underestimation of curvature, especially near diagnostic thresholds, underscoring the need for standardised protocols or digital tools to improve measurement reliability [15].

2.3. Lumbar Disc Herniation

LDH is a pathological condition characterized by the displacement of the nucleus pulposus through the annulus fibrosus, typically in a posterior or posterolateral direction. This herniation leads to narrowing of the vertebral canal or neural foramina, resulting in mechanical compression of the thecal sac or adjacent nerve roots [3]. The incidence of this condition is approximately 5 to 20 cases per 1,000 adults per year. The majority of herniations occur in the lower lumbar spine, with 95% of cases located at the L4-L5 and L5-S1 levels. Herniations at higher levels are more frequently observed in individuals over the age of 55 [4].

Although LDH is often idiopathic in origin, it may also result from trauma or from age-related degenerative changes. Several risk factors are known to predispose individuals to this condition, including obesity, smoking, diabetes, connective tissue disorders, and genetic predisposition. In many middle-aged and elderly patients, LDH is associated with spondylosis, contributing to symptomatic nerve compression due to reduced space available for neural structures. Similarly, in younger patients with congenital spinal canal stenosis, even minor herniations may produce significant clinical symptoms because of the preexisting structural limitation [5]. According to Skaf et al 2011, age and sagittal spinal curvature influence the level at which lumbar disc herniation occurs. Their retrospective study showed that increasing age and changes in the lumbar lordotic angle were associated with herniations occurring more frequently at lower lumbar levels. These findings suggest that the morphology of lumbar curvature alters mechanical loading patterns, affecting which discs are most susceptible to herniation, and highlight the need to consider both demographic and anatomical factors when evaluating disc pathology [16].

Clinically, herniated disc material may compress the posterior longitudinal ligament and generate local inflammation, producing axial lumbar pain. Radicular symptoms arise when the disc compresses the thecal sac or nerve roots, causing inflammation and ischaemia. The annulus fibrosus is most vulnerable in the posterolateral region due to its thinner structure and limited support from the posterior longitudinal ligament, making posterolateral herniations the most likely to provoke nerve compression and neurological deficits [5].

Persistent or inadequately managed LDH can initiate a broader degenerative cascade. Genevay and Atlas 2010 describe how progressive disc height loss alters load distribution and facet joint articulation, promoting hypertrophy of the ligamentum flavum and facet joints. These changes reduce canal and foraminal dimensions, ultimately contributing to acquired lumbar spinal stenosis, characterised by neurogenic claudication and radicular pain [17].

Degenerative and biomechanical alterations may also predispose individuals to adjacent segment disease (ASD), in which segments adjacent a degenerated or fused disc undergo accelerated degeneration. Lee et al. 2009 identified pre-existing facet degeneration and sagittal

malalignment as major risk factors, noting that altered load distribution after lumbar fusion increases stress on adjacent levels and accelerates their deterioration. Importantly, the authors suggest that longstanding disc pathology may produce similar biomechanical consequences even without surgery, an implication that is particularly relevant for the motivation of this study [18].

Further expanding on ASD, Louie et al. (2020) proposed an aetiology-based classification in which symptomatic disc degeneration, spinal stenosis, and radiculopathy constitute the main clinical manifestations. Their findings emphasise its multifactorial nature, involving surgical mechanics, pre-existing degeneration, and individual anatomical variability [19]. Tsuji et al. 2021 provided complementary diagnostic criteria integrating radiographic findings with patient symptoms, reporting that many patients who undergo lumbar fusion later develop new stenosis or herniation at adjacent levels, reinforcing the concept that biomechanical disruption at one spinal segment can promote degeneration elsewhere in the lumbar spine [20].

2.3.1. Characterization of Lumbar Disc Herniation

A critical aspect of LDH evaluation is its craniocaudal orientation, which can be classified as ascending, descending, or at the disc level. Ascending LDH refers to disc material that migrates superiorly relative to the disc space, whereas descending LDH involves inferior migration. In contrast, herniation at the disc level describes a protruded or extruded fragment confined within the vertical height of the IVD space, without significant cranial or caudal displacement. This typically results in direct compression of the adjacent nerve roots at the level of origin [21].

These variations in the herniation trajectory have significant implications for clinical presentation, nerve root involvement, and therapeutic decision-making. While MRI remains the gold standard for characterising disc morphology and soft-tissue involvement, CT can provide complementary information in specific contexts, particularly for assessing bony structures, calcified herniations, or when MRI is contraindicated. Accurate MRI-based diagnosis, combined with a thorough understanding of these morphological and topographical distinctions, is essential for optimizing patient outcomes and guiding evidence-based management strategies [21].

Herniation can also be classified by topographic position in sagittal or coronal planes, including central, paracentral, foraminal, or extraforaminal locations. The subarticular (paracentral) zone corresponds to the right or left region of the vertebral canal. Anatomical zones are illustrated in axial and coronal projections (Figure 2.4) [22].

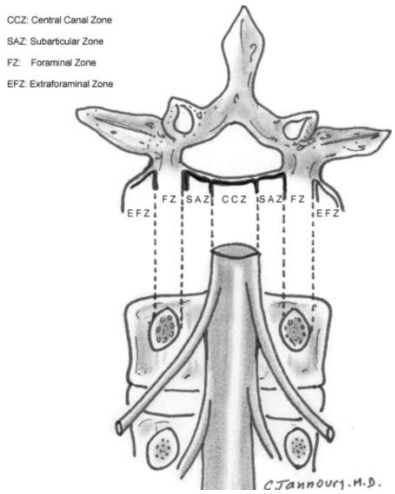

Figure 2.4 - Topography of LDH. Adapted from Oh et al 2017 [23].

Based on their morphological appearance and imaging characteristics, herniations can be further classified into three subtypes (Figure 2.5):

- ***Protrusion***: When the greatest distance between the edges of the disc material outside the disc space is less than the distance between the edges of the base of that material. Anatomically, this is a focal displacement with minimal disruption of the annulus fibrosus and an intact posterior longitudinal ligament [21], [22].
- ***Extrusion***: Identified when, in at least one plane, the distance between the edges of the disc material beyond the disc space exceeds the base width, or when there is no continuity between herniated and in-disc material. This indicates a full-thickness disruption of the annulus fibrosus [21], [22].
- ***Extrusion with sequestration***: Represents a type of extrusion where the extruded disc material has no continuity with the disc of origin [21], [22].

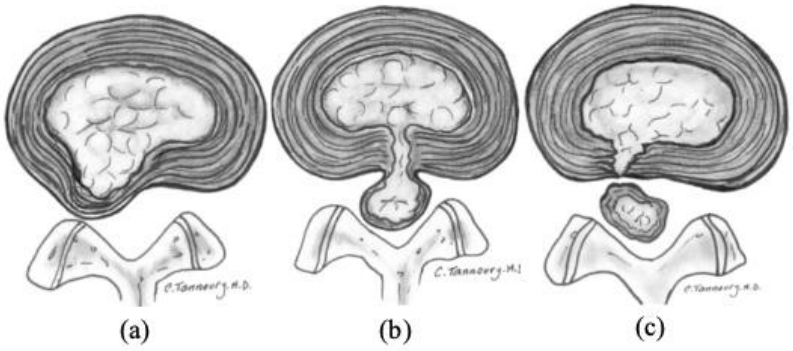

Figure 2.5 - Axial representations of the types of herniated discs. (a) Representation of protruded disc; (b) Representation of extruded disc; (c) Representation of extruded with sequestration disc. Adapted from Fardon et al. 2014 [22].

To complement this morphological classification, the Pfirrmann classification system is widely utilized to assess IVD degeneration. This system evaluates MRI signal intensity, disc structure, nuclear-annular distinction, and disc height. It grades degeneration from I (normal) to V (advanced degeneration). The grading criteria are summarized in Table 2.1, and the associated imaging examples, following the Pfirrmann Classification from Grade I (healthy) to Grade V (collapsed), are provided in Figure 2.6 [21], [23].

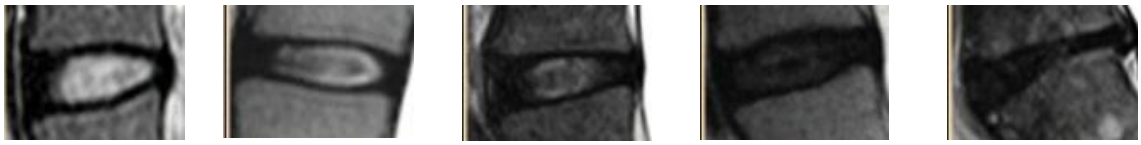

Figure 2.6 - Visual representation of Pfirrmann Classification System. Adapted from Kushchayev et al 2018 [21].

Table 2.1 - Parameters of Pfirrmann Classification System. Adapted from Oh et al 2017 [23].

Grade	Structure	Distinction of NP and AF	Signal Intensity	IVD Height
I	Homogeneous, bright white	Clear	Hyperintense, isointense to cerebrospinal fluid	Normal
II	Inhomogeneous with/without horizontal bands	Clear	Hyperintense, isointense to cerebrospinal fluid	Normal
III	Inhomogeneous, grey	Unclear	Intermediate	Normal to slightly decreased
IV	Inhomogeneous, grey to black	Lost	Intermediate to hypointense	Normal to moderately decreased
V	Inhomogeneous, black	Lost	Hypointense	Collapsed

2.3.2. Treatment Plan for LDH

Initial management of LDH with radiculopathy focuses on conservative therapies, including physiotherapy and non-steroidal anti-inflammatory drugs (NSAIDs), since approximately 70-80% of patients experience symptom resolution within 6-12 weeks without surgical intervention [22], [24].

For patients with persistent symptoms beyond six weeks, fluoroscopy-guided epidural steroid injections (ESIs) may provide short-term relief by reducing inflammation around the affected nerve root, though their long-term efficacy remains controversial [25].

Surgical intervention is generally considered appropriate for patients who exhibit persistent radicular pain, accompanied by physical signs of nerve root irritation, such as motor or sensory deficits or a positive straight leg raise test, and who have not responded to 4 to 6 weeks of conservative treatment [26]. Surgery becomes imperative in cases of progressive neurological deficits (for example, motor weakness) or cauda equina syndrome [27].

Minimally invasive surgical (MIS) approaches, including endoscopic and microsurgical discectomy, have gained popularity due to reduced blood loss, less extensive tissue dissection

and shorter hospitalization. However, concerns persist regarding safety due to the limited working space, which may increase technical complexity and risk of dural tears [28], [29], [30].

The gold standard surgical treatment is open microdiscectomy, which provides excellent visualization and decompresses the affected nerve root via a small laminectomy, while preserving surrounding tissue as much as possible [27]. Early surgical intervention (4 to 6 weeks of conservative treatment) is associated with faster functional recovery, although delayed surgery does not necessarily lead to worse long-term outcomes [31].

2.4. Diagnostic Methods and Analysis

Accurate diagnosis of LDH is essential for guiding appropriate clinical management and improving patient outcomes. Given the (sometimes) inconsistent correlation between anatomical abnormalities and clinical symptoms, a comprehensive diagnostic approach typically involves the integration of a detailed clinical examination with advanced imaging techniques. This section outlines the primary imaging modalities used in the evaluation of LDH, emphasizing their diagnostic capabilities, advantages, limitations, and clinical relevance.

2.4.1. Imaging Modalities

Diagnostic imaging plays a fundamental role in the evaluation of patients presenting with back and/or leg pain, especially when assessing for nerve root compression caused by LDH, spinal stenosis, or cauda equina syndrome. Commonly used imaging techniques include MRI, CT and plain radiography (X-ray) that's mainly used to evaluate the cause of the herniation [32].

Despite the widespread use of these modalities, the diagnostic accuracy in LDH remains somewhat variable, with frequent discrepancies observed between patients' clinical symptoms and MRI findings (the best image modality for the diagnosis). This discordance can complicate the diagnostic process and influence therapeutic decision-making [32].

2.4.1.1. MRI

MRI is currently regarded as the imaging modality of choice for evaluating LDH due to its avoidance of ionizing radiation and its superior soft tissue contrast [32]. With a diagnostic accuracy of approximately 97%, MRI is the most sensitive tool available for detecting herniated discs, particularly because of its ability to identify increased T2-weighted signal intensity in the posterior 10% of the disc [3].

Additionally, MRI demonstrates higher inter-observer reliability compared to other imaging modalities and is more effective than CT in differentiating between inflammatory, malignant, or infectious causes of LDH. It is typically indicated early in the diagnostic workup, within the first weeks, for patients presenting with severe pain, motor deficits, or clinical signs suggestive of cauda equina syndrome [3].

The administration of intravenous paramagnetic contrast agents can further enhance anatomical detail, particularly in patients with prior lumbar surgery. MRI also provides a superior

assessment of disc morphology related to CT. Moreover, it allows for a more accurate evaluation of foraminal involvement and anterior disc herniations, findings that are critical for effective surgical planning and procedural strategy [33], [34].

In Çetin et al. 2023, an MRI-based study, the authors compared 385 patients with and without LDH, evaluating disc space height at the L4-L5 level and grading degeneration using the Pfirrmann classification. Patients with herniation exhibited significantly reduced disc height across anterior, middle, and posterior regions, and they also showed higher Pfirrmann grades, indicating more advanced degeneration. While age was higher in the herniated group, gender was not a significant factor. These findings suggest that decreased disc height and advanced degeneration are closely linked with disc herniation, and that quantitative MRI measurements can provide reliable markers for differentiating between healthy and diseased disc levels [35].

In Tunset et al. 2013, a methodological study introduced and validated a quantitative MRI-based approach for measuring lumbar IVD structures, including disc height, disc bulge, and dural sac diameter. Sixteen patients with herniation and sixteen without were included, and measurements were performed repeatedly by different raters to evaluate intra- and inter-rater agreement. The results demonstrated good to excellent reliability for most parameters, with intraclass correlation coefficients generally above 0.8. Some structures with less clearly defined boundaries, such as the dural sac or small disc protrusions, showed slightly lower reliability. Overall, the study confirmed that quantitative imaging methods can provide reproducible measures of lumbar disc morphology, offering advantages for both clinical monitoring and research over traditional qualitative approaches [36].

2.4.1.2. CT

CT is widely available and frequently used to assess spinal morphology, particularly vertebral abnormalities. Although it can indicate disc pathology through associated structural changes, its limited soft-tissue contrast makes MRI the preferred method for diagnosing disc herniation [32]. CT is highly sensitive for detecting bony structures, calcified herniations, and destructive bone lesions, but its ability to visualise nerve roots is limited, reducing its usefulness in evaluating radiculopathy [3]. Compared with MRI, CT is faster, more accessible, and generally less costly, although its use is restricted by exposure to ionising radiation, particularly in younger or repeatedly imaged patients [31]. While CT provides clear anatomical information and is effective for identifying large herniations, differentiating protrusions from extrusions can be difficult without sagittal reconstructions, and small herniations may be missed, leading to potential diagnostic inaccuracies [33].

2.4.1.3. X-Ray

Although plain radiography is not indicated for the diagnosis of LDH, it remains a useful initial imaging modality for excluding other spinal pathologies such as spondylolisthesis, instability and for providing baseline anatomical information to guide further evaluation or surgical planning.

Flexion-extension lateral views can help assess segmental stability when instability is suspected [37].

2.4.2. Modelling and Segmentation

Manual interpretation of medical image series, often comprising thousands of slices, is time-consuming and prone to error. To improve efficiency and diagnostic accuracy, computer-aided diagnosis (CAD) has emerged as a key focus in medical imaging. CAD systems leverage computer vision to direct radiologist's attention toward diagnostically significant regions, offer quantitative assessments of suspect tissue, automate anomaly segmentation, and facilitate retrieval of similar cases from image databases. 3D computational analysis of biomedical textures supports large-scale image screening and increasingly higher spatial resolution imaging. As datasets become more complex, reliable computational tools are essential not only for interpretation but also for downstream modelling workflows, including biomechanical simulation. With these advancements, robust computational tools are essential to manage and interpret the increasing complexity and volume of imaging data. [38].

Alongside image segmentation, finite element analysis (FEA, a numerical method employed in the simulation of tissues or implants, with frequent application in spine biomechanics and degeneration) has become a key computational approach in spinal modelling. It's a non-invasive numerical tool used to simulate and analyse the mechanical behaviour of the lumbar spine in a virtual environment [39], allowing the assignment of material properties to specific geometric regions for detailed analysis of stress distribution, displacement, and load transfer. These capabilities facilitate the optimization of prosthetic geometries, evaluation of surgical interventions, and design of more effective spinal implants. Compared to in vivo methods, FEA offers improved safety, cost-efficiency, and experimental control [39], [40].

CAD and FEA are fundamentally dependent on accurate image segmentation [41]. Deep learning-based methods, particularly convolutional neural networks (CNNs) such as cascaded 3D U-Nets, have achieved high performance in CT and MRI spine segmentation, with mean Dice similarity coefficients exceeding 95% [40], [42]. Despite this progress, fully automated segmentation-to-mesh pipelines remain challenging due to vertebral labelling, anatomical variability, and material property assignment [42]. To address these limitations, recent architectures have integrated CNNs with Transformer-based modules. For instance, SymTC, leverages local CNN features and global Transformer context to improve lumbar spine MRI segmentation, outperforming state-of-the-art methods in distinguishing vertebrae and IVDs. However, its evaluation was restricted to small, curated datasets and lacked external clinical validation [43].

Beyond anatomical segmentation, automated quantification and diagnostic classification of spinal pathology have gained increasing attention. The study by Yilihamu et al. proposed a deep learning system for quantification and classification of LDH on axial MRI. Their model automatically identified IVDs, measured herniation size, and categorized pathology types with

performance comparable to expert radiologists. This extension from pure segmentation to quantitative and diagnostic tasks demonstrates the potential of AI systems to reduce variability and support standardized workflows. However, the evaluation was retrospective and based solely on imaging features, without incorporating biomechanical or functional data, leaving its clinical applicability uncertain [44].

Recent advancements have improved the automation and accuracy of FEM workflows, enabling faster generation of patient-specific models and more reproducible biomechanical analyses [44]. Although such developments highlight the potential of integrating imaging and physics-based modelling, they remain outside the scope of the present work [45].

Established biomechanical evidence shows that alterations in a single lumbar motion segment, such as those produced by disc herniation, can modify load transfer, intradiscal pressures, and mobility patterns in adjacent levels, potentially accelerating degenerative changes [46]. Still, prior research has shown that quantitative information extracted directly from MRI, particularly disc shape and signal intensity profiles, can provide clinically meaningful markers of disc condition. In this context, Matos 2021, demonstrating that variations in T2-weighted signal intensity are strongly linked to disc health and degeneration. These results strengthen the diagnostic value of MRI-derived intensity-based parameters, supporting their use in studies that aim to assess how lumbar pathology, such as disc herniations, may influence the condition of adjacent discs [47].

CHAPTER 3

METHODS AND MATERIALS

This chapter presents the methodological framework of the study, including the study design, sample characteristics, and the procedures used to ensure data validity, reliability, and ethical compliance. It also outlines the clinical and imaging assessment methods and the process of data organisation and analysis.

3.1. Study Design

This study follows an observational cross-sectional design with a sample of 26 patients. All participants were selected according to the inclusion and exclusion criteria pre-established in the study protocol, as presented in Tables 3.1 and 3.2.

Table 3.1 - Inclusion criteria for this study.

	Inclusion Criteria
(1)	Age between 18 and 80 years
(2)	LDH at L4-L5/L5-S1 or both
(3)	Diagnosis based on clinical and imaging criteria (prolapse, protrusion, extrusion or sequestration)
(4)	Radicular pain with evidence of nerve-root irritation with a positive nerve-root sign
(5)	Ability to provide consent

Table 3.2 - Exclusion criteria for this study.

	Exclusion Criteria
(1)	Previous spine surgery
(2)	Cauda equina syndrome
(3)	Neurological disease that might interfere with gait
(4)	Own orthopaedic conditions that significantly disrupt gait, as per investigator judgement
(5)	Severe comorbidity
(6)	Vertebral fractures
(7)	Spine infection or tumour
(8)	Pregnancy
(9)	Dementia or any other form of cognitive impairment
(10)	Inability to provide informed consent
(11)	Spondylolisthesis and/or spinal instability

3.2. Sample

To establish the database, several demographic variables were collected, including age, sex, weight, height, and body mass index, together with information on the herniation level and the Pfirrmann grade across the entire lumbar spine. The sample is divided into three subgroups according to the herniation level. In table 3.3 are represented the clinical parameters of group H. All clinical parameters are presented in Appendix A.

- Group H.1 consists of patients with a herniation at the L4-L5 segment;
- Group H.2 includes patients with a herniation at the L5-S1 level;
- Group H.3 comprises patients with two herniations in the last two lumbar segments.

Table 3.3 - Clinical parameters of Group H.

	Nº of patients		Mean Age	Mean Weight	Mean Height	Mean BMI
	Male	**Female**	**(years)**	**(kg)**	**(cm)**	**kg/m²**
Group H.1	6	1	52,6	83	170,43	29
Group H.2	3	12	47,6	72,7	164,1	27
Group H.3	6	1	49	89,1	169,5	30,2

To explore potential associations between demographic and lumbar spine degeneration using Pfirrmann Classification, patients were divided into age intervals. This stratification enabled the analysis of whether younger individuals exhibited unexpectedly advanced degeneration or a higher number of herniated discs. In table 3.4 are represented the clinical parameters of group A.

- Group A.1 includes patients aged between 21 and 40 years;
- Group A.2 consists of patients aged between 41 and 60 years;
- Group A.3 comprises patients aged between 61 and 80 years.

Table 3.4 - Clinical parameters of Group A.

	Nº of patients		Mean Age	Mean Weight	Mean Height	Mean BMI
	Male	**Female**	**(years)**	**(kg)**	**(cm)**	**kg/m²**
Group A.1	5	2	30.3	73.4	174,1	23.9
Group A.2	7	7	52.4	82.6	164,1	30.1
Group A.3	3	2	67.8	81.6	165.4	30,0

In parallel, patients were categorized according to BMI classification. This approach aimed to assess whether higher BMI values were associated with increased degenerative changes or with the presence of more herniated levels. BMI categories were defined according to the standard World Health Organization (WHO) adult classification (Table 3.5) [48]. In table 3.6 are represented the clinical parameters of Group B.

- Group B.1 includes patients with an ideal BMI;
- Group B.2 consists of patients who are overweight;
- Group B.3 comprises obese patients, encompassing all three obesity grades (I, II, and III).

Table 3.5 - BMI Classification, from [48].

BMI Classification	
Below Weight	<18.5 Kg/m²
Normal Weight	18.5 - 24.90 Kg/m²
Overweight	25 - 29.90 Kg/m²
Obesity Grade I	30 - 34.90 Kg/m²
Obesity Grade II	35 - 39.90 Kg/m²
Obesity Grade III	> 40 Kg/m²

Table 3.6 - Clinical parameters of Group B.

	Nº of patients		**Mean Age**	**Mean Weight**	**Mean Height**	**Mean BMI**
	Male	**Female**	**(years)**	**(kg)**	**(cm)**	**kg/m²**
Group A.1	6	2	43.5	68	171,3	23.1
Group A.2	5	5	49.1	75.8	165,7	27.4
Group A.3	4	4	55.6	97	164.6	32.4

3.3. Study Procedures

The initial phase of the study involved the development of a Study Protocol, along with the collection of all necessary documentation for submission to the Research Board and Ethics Committees of Grupo Luz Saúde. Approval from these entities was obtained to ensure adherence to ethical standards and compliance with relevant international, European, and national regulations, including the Declaration of Helsinki and the Charter of Fundamental Rights of the European Union.

Following ethical clearance, patients diagnosed with LDH were recruited from the outpatient clinic of the Neurosurgery Department at HLS, between April 2024 and August 2025. Each patient received an informed consent form, which provided a thorough explanation of the study, including its duration, potential risks, confidentiality measures, and the rights of participants.

Once eligibility was confirmed and written consent obtained, each participant was assigned a unique study identification number for data protection purposes.

3.3.1. Clinical Assessment

The choice of initial diagnostic imaging reflects an inherent bias related to the specialized neurosurgical setting, where patients are typically referred to, due to severe pain or prolonged

symptom duration, often in consideration for more complex interventions such as surgery. In these cases, neurosurgeons tend to prefer MRI over CT scans as the first-line imaging modality, given their superior capability in visualizing IVDs. The study included only patients who had MRI, as this was the imaging modality used for analysis.

Following imaging, the neurosurgeon determines the treatment approach based on clinical judgment and standard clinical practices. This decision considers the presence, intensity, and duration of both LBP and radicular symptoms, as well as the confirmation of LDH on imaging. Patients presenting with LDH and mild symptoms are assigned to a conservative treatment group, whereas those with moderate to severe and/or persistent symptoms are directed toward surgical management.

3.3.2. Data Collection

The data was recorded in a database (DB) developed exclusively for the study, which included: (1) demographic data (age and sex); (2) standardized clinical evaluation (including also height, weight and BMI) and (3) treatment group.

The diagnosis of LDH was established based on MRI findings for all patients. Most MRI scans were obtained using a GE 3T MRI system (Signa Architect AIR), except for six cases whose scans had been performed at other clinics or hospitals.

During MRI acquisition, patients were positioned in the supine position according to the standard lumbar spine imaging protocol.

After image acquisition, a set of quantitative and qualitative parameters was extracted from the MRI scans. Their selection was based on a previous study from our research group and refined through discussions with neurosurgeons, radiologists, and imaging technicians at HLS [49]. A detailed description of the quantitative parameters, including imaging plane, slice location, measurement units, and extraction procedures, is provided in that study [49]. The parameters extracted are listed in Table 3.7.

Table 3.7 - Measured Parameters from MRI.

Measured Parameters	
IVD Height	Intervertebral Angle (Sagittal Plane)
Vertebrae Height	Intervertebral Angle (Coronal Plane)
Lateral Deviation	LLA

All image measurements were carried out using Carestream Vue Motion (Onex Corporation, USA), which allows for high precision. Each parameter was manually measured three times for each patient, with time intervals between measurements to minimise potential bias due to learning or visual adaptation.

The heights of the IVDs and vertebrae did not always align along with the same orientation, as the inclination of the spine naturally varies between different vertebral levels in

each patient. Additionally, because not all patients had a coronal plane available, multiplanar reconstruction (MPR) was used to obtain the necessary views for certain measurements. MPR is a software technique that enables the computational reconstruction of three-dimensional MRI volume data into multiple planes for visualization on a 3D workstation, allowing the user to view and measure structures in any desired orientation [50]. While MPR made it possible to visualize planes that were not initially available, this process reduced the overall image quality, which may have influenced some of the subsequent measurements.

The lateral deviation of the lumbar spine represents the curvature angle that causes a displacement of the central plane of each vertebra, resulting in a manual adjustment of the vertebral midplane during the measurement of height-related parameters, which would otherwise present more disparate values. The measurement of this parameter is performed using MPR reconstruction from the midplane of L1, in the coronal plane, with the aid of a reference line (visible in Figure 3.1, shown in blue in the sagittal plane), also projected onto the coronal plane to ensure that the L1 midplane is maintained throughout the entire lumbar spine. Subsequently, the midplane of L4 (the last vertebra visible in all ten patients within the L1 midplane) is determined and connected to the midplane of L1. Finally, the angle between these two markings is measured to assess whether any significant lateral deviation is present. This measurement was assessed in 10 patients, corresponding to the cases in which this parameter could be obtained. In Figure 3.1 is represented an example of how the measurement was made in patient 8.

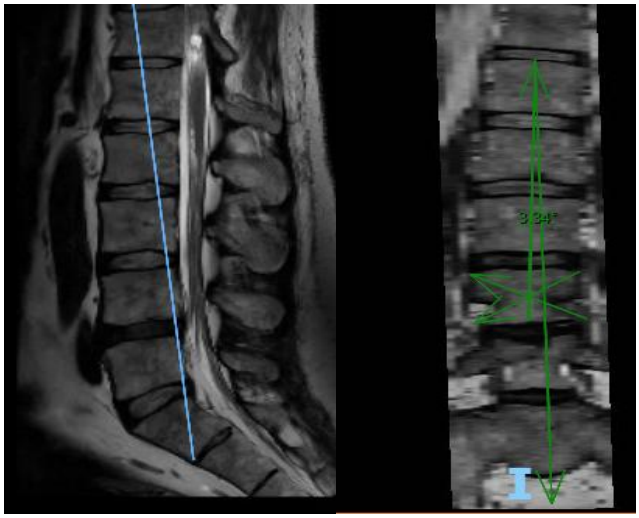

Figure 3.1 - Measurement of Lateral Deviation (angle) of Patient 8 using MPR reconstruction.

After obtaining the three measurements, their average value was calculated. The deviation from this average value was then determined to evaluate the reliability and consistency of the results (Eq. 3.1).

$$Deviation = \frac{Average - Rep_x}{Average} \times 100 \tag{3.1}$$

This formula was applied as an adaptation of the standard percentage error specifically to calculate the deviation of each measurement from the mean, rather than from a true or reference value. It was applied to each of the three repetitions, followed by the calculation of the average deviation across all repetitions. The result illustrates the dispersion of the data relative to the mean, that is, the greater the variation among individual measurements, the higher the deviation.

In addition, Equation 3.2 was applied to the IVD height, intervertebral angle, and vertebral height only in anterior and posterior heights. This equation was used to assess the difference in height between two consecutive levels (difference between levels, DBF), considering the value of the preceding segment.

$$DBF = -\frac{Lower\ level - Upper\ level}{Lower\ level} \times 100 \tag{3.2}$$

When applying Equation 3.2, a positive value indicates an increase in intervertebral disc height when moving from one segment to the next inferior level, whereas a negative value represents a decrease. This equation is applied both to the measurements obtained in this study and to those reported in the literature, allowing comparison of whether the observed loss in disc height results from lumbar spine degeneration or whether herniation in the lower segments has any effect on the height of adjacent discs. When applied to the posterior intervertebral disc heights at levels L4-L5 and L5-S1 in Patient 1, the analysis shows that:

$$DBF\ (example) = -\frac{5.09 - 6.58}{5.09} \times 100 = 23\% \tag{3.3}$$

In Figure 3.2, it is possible to see a difference in the height in the measurements (anterior and posterior) of the disc used for the analysis.

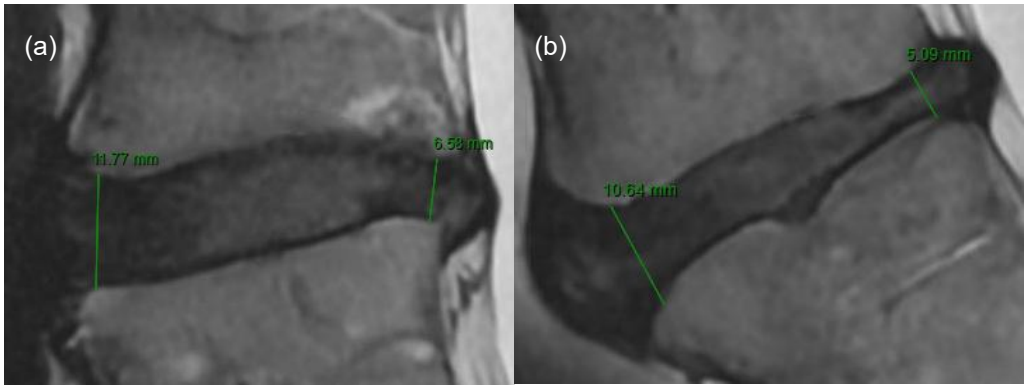

Figure 3.2 - Intervertebral Height of Patient 1; a) L4-L5 measurements; b) L5-S1 measurements.

3.3.3. Qualitative Assessment of Disc Degeneration

In addition to the quantitative MRI measurements described above, disc degeneration was assessed using the Pfirrmann classification, which provides a qualitative evaluation of disc structure and signal intensity. This parameter was also extracted manually.

This classification was applied to all lumbar levels from Grade I (healthy) discs to Grade V (severe degeneration) discs, allowing the characterization of the degeneration of the spine beyond the herniated segment, in figure 3.1 is represented an example of Pfirrmann Classification across the lumbar spine. Since disc herniation and disc degeneration refer to distinct clinical and anatomical processes, the inclusion of Pfirrmann grading provided an independent evaluation of the degenerative status of adjacent and non-herniated levels, not influenced by the variability associated with quantitative measurements.

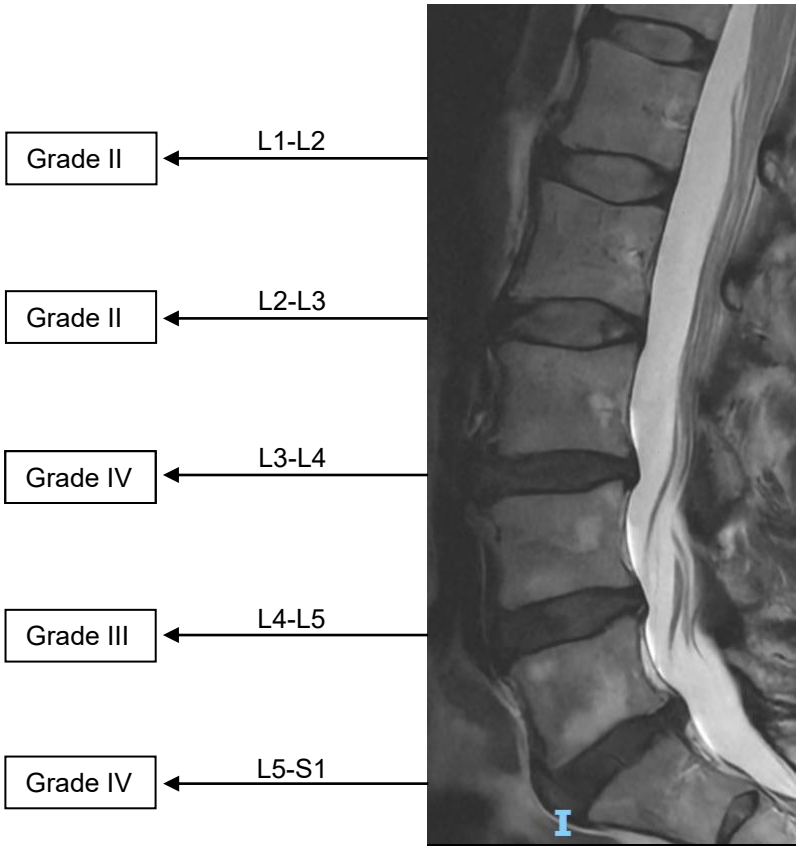

Figure 3.3 - MRI imaging of LLA in sagittal plane of patient 11.

CHAPTER 4

RESULTS AND DISCUSSION

Following the methodological procedures outlined in the previous chapter, this section presents and interprets the results obtained during the experimental process. These results form the foundation for the critical discussion and final conclusions presented in Chapter 5.

4.1. Characterization of Measurements Quality

In this study, both inter- and intra-observer analyses were performed. The lateral deviation parameter was only assessed through intra-observer analysis, as it was only evaluated by the author, whereas all remaining parameters underwent inter-observer analysis by comparing the values collected in this study with those reported previously by other researcher of our group, using the same dataset and methodology [49].

The parameters evaluated included IVD and vertebral body height, intervertebral angles in the sagittal and coronal planes, and Pfirrmann classification. The average deviation for each parameter was calculated using Equation 3.1.

The intra-observer deviations for the measurements were 1% to 6% for IVD height and 1% to 2% for vertebral body height. The intervertebral angle showed greater intra-observer variability, with deviations between 10% and 22% in the sagittal plane and 10% to 43% in the coronal plane. The LLA presented an intra-observer deviation of 2%.

Considering these results, variables with deviations below 10% were regarded as reliable and included in subsequent analyses. Although intervertebral angles presented higher deviations, they were still analysed due to their anatomical and clinical relevance.

For inter-observer reliability, a comparison with the study by Mendes revealed similar patterns, with deviations of 1% to 4% for disc height, 1% to 2% for vertebral height, 7% to 18% for the sagittal intervertebral angle and 16% to 43% for the coronal plane. The LLA presented a 2% deviation [49].

These results reinforce that several manual measurements lack sufficient reliability for conclusive interpretation, emphasising the need for more precise approaches, such as computational tools for automated parameter extraction. Consequently, while intervertebral angle measurements were included, no definitive conclusions were drawn from these values.

The average deviation was also calculated for the lateral deviation parameter, which was not included in the previous study. This value, presented in the Appendix B, corresponded to a mean deviation of 9%. However, because this parameter could be extracted from only 10 patients, further statistical analysis was not feasible.

4.2. Characterization of Intervertebral Height

Since LDH is associated with the rupture of the annulus fibrosus of the intervertebral disc, it is implicitly understood that a loss of intervertebral height occurs. Therefore, intervertebral disc height is the first parameter to be analysed, as it reflects the degree of disc degeneration and structural compromise resulting from the herniation process. The average values of the intervertebral height are in Appendix B.

4.2.1. IVD Height: Herniated vs Normal

The IVD heights of the patients included in the present study were analysed and compared with a study involving 171 normal individuals (84 men and 87 women) aged between 12 and 80 years. The results confirmed that lumbar IVD height generally increases progressively from the upper to the lower levels, which is consistent with the idea that lower lumbar segments, being subjected to greater mechanical loads, tend to exhibit larger disc heights, possibly as an adaptive structural characteristic rather than a direct consequence of load itself. It was also observed that, at all levels, anterior disc height is greater than posterior height, a characteristic associated with the lumbar lordotic curvature. Between L4-L5 and L5-S1, a slight decrease in height was noted (mainly in the posterior height), which may be associated with the transition to the sacrum and the increase in lumbar lordosis typically observed at these levels. Although some variations were identified with advancing age, changes in disc height were not consistent, suggesting that aging influences disc morphology in a non-uniform manner along the lumbar spine [51].

From Table 4.1, the differences in anterior and posterior intervertebral disc height among patients with LDH can be observed and compared to reference values in Table 4.2.

Table 4.1 - Average anterior and posterior IVD height in LDH patients.

	L1-L2 to L2-L3		L2-L3 to L3-L4		L3-L4 to L4-L5		L4-L5 to L5-S1	
	A	**P**	**A**	**P**	**A**	**P**	**A**	**P**
Group H.1	8%	15%	5%	-3%	13%	19%	37%	-10%
Group H.2	15%	13%	16%	5%	19%	3%	-7%	-23%
Group H.3	12%	12%	15%	8%	11%	-8%	-3%	-8%

A - Anterior; P - Posterior.

Table 4.2 - Reference values of anterior and posterior IVD height [51].

	Reference Values for IVD Height			
	L1-L2 to L2-L3	**L2-L3 to L3-L4**	**L3-L4 to L4-L5**	**L4-L5 to L5-S1**
Anterior	12%	11%	13%	6%
Posterior	9%	5%	5%	-14%

In Group H.1, anterior disc height aligns with the reference value of increasing toward the lower lumbar spine, except for the last lumbar segment that demonstrated a substantial increase compared with reference values. This increase at L5-S1 was greater than that observed at

adjacent levels and exceeded the change reported in previous literature. Posterior disc height showed a slight increase at the herniated level, while both superior and inferior adjacent segments exhibited reductions compared with reference patterns. The reduction in posterior height was more evident at the superior adjacent segment than at the lowest level. These results suggest that when herniation occurs at L4-L5, the disc height variation is not restricted to the symptomatic level alone. Instead, there is an associated pattern of anterior height increase at the immediately inferior segment and posterior reductions adjacent to the herniated disc.

Group H.2 demonstrated a decrease in anterior disc height at the herniated segment. In contrast, adjacent discs displayed anterior increases relative to the reference values. Posterior disc height showed an overall increasing tendency across levels, but a pronounced reduction was present at the herniated level, which was approximately double the reduction suggested by the reference values for this segment. The pattern in this group indicates that disc height alterations appear most pronounced at the herniated level, with both anterior and posterior measures showing deviations from the expected height. Adjacent levels demonstrated increases, which may be associated with morphological changes.

In Group H.3, anterior disc height was reduced at the lowest segment, whereas disc heights at more upper levels, including the herniated L4-L5, showed values comparable to reference data. Posterior disc height increased at all superior adjacent levels but decreased at both herniated segments. The reduction at L4-L5 occurred despite reference values indicating an expected increase at this level. At L5-S1, a reduction was present but smaller than expected for that segment. This pattern suggests distinct height behaviour in the presence of two-level herniation, with greater consistency in the more superior discs and localized reductions at herniated levels.

Across all groups, the L2-L3 level consistently showed an increase in disc height compared with the reference values, suggesting a recurring pattern at this mid-lumbar segment regardless of herniation level. Adjacent levels tended to demonstrate variations in both directions, but posterior disc height showed a clearer tendency toward increases at superior adjacent segments, while the herniated discs generally presented reduced posterior height.

Anterior disc height presented a wider range of variation; however, when alterations were present, inferior adjacent segments showed a tendency toward increases, particularly when the herniation was located at L4-L5.

The results indicate that LDH at the lowest lumbar levels is associated with measurable alterations in IVD height at both the affected segment and the adjacent levels. Although patterns differ between groups, a recurring result is that herniated levels tend to show reductions in posterior disc height, while superior adjacent discs frequently exhibit increases. Anterior disc height changes were less uniform, though in several cases, inferior adjacent discs increased in height.

4.2.2. IVD Height vs Age

An analysis of anterior and posterior IVD heights as a function of age was performed for all patients, separated into the three patient groups. Tables 4.3, 4.4, and 4.5 present the trend line of each linear regression for the patients in each group, divided by spinal level and by height (anterior or posterior).

The linear regression analysis was performed to assess the relationship between IVD height and patient's age (Figures 4.1, 4.2 and 4.3). The slope of the regression line indicates how much IVD height changes for each unit increase in age, reflecting the strength and direction of their association. The coefficient of determination (R^2) quantifies how well patient height explains the variability in IVD height, with higher values indicating a stronger linear relationship.

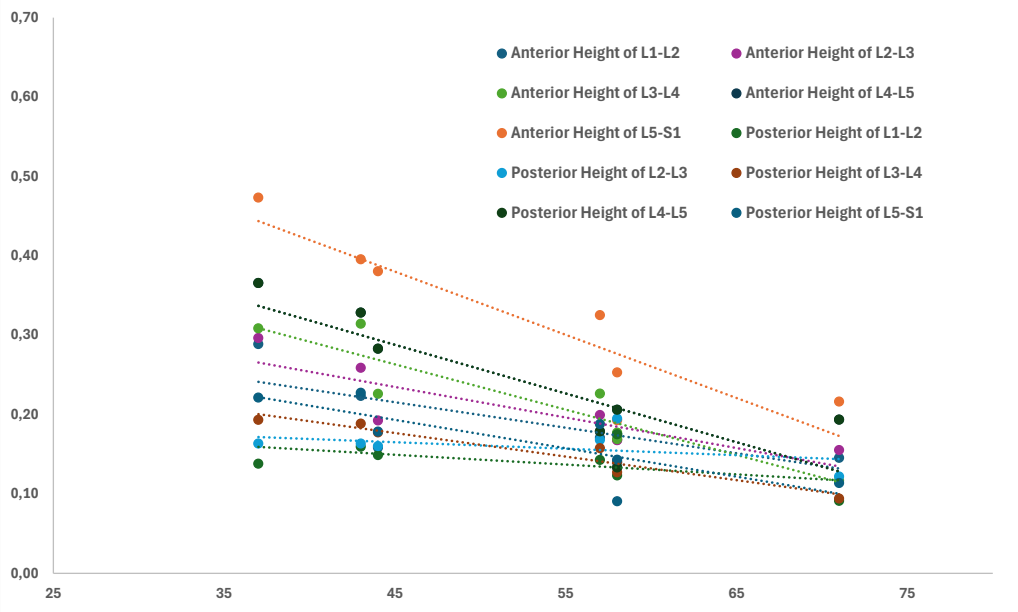

Figure 4.1 - Linear Regression between IVD height and Age in Group H.1.

Table 4.3 - Correlation between IVD height and Age in Group H.1.

Group H.1	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1-L2	-0,0032	0,5823	-0,0012	0,2887
L2-L3	-0,0038	0,7318	-0,008	0,1742
L3-L4	-0,0057	0,8442	-0,003	0,9338
L4-L5	-0,0061	0,7074	-0,0061	0,7074
L5-S1	-0,008	0,8133	-0,0036	0,6452

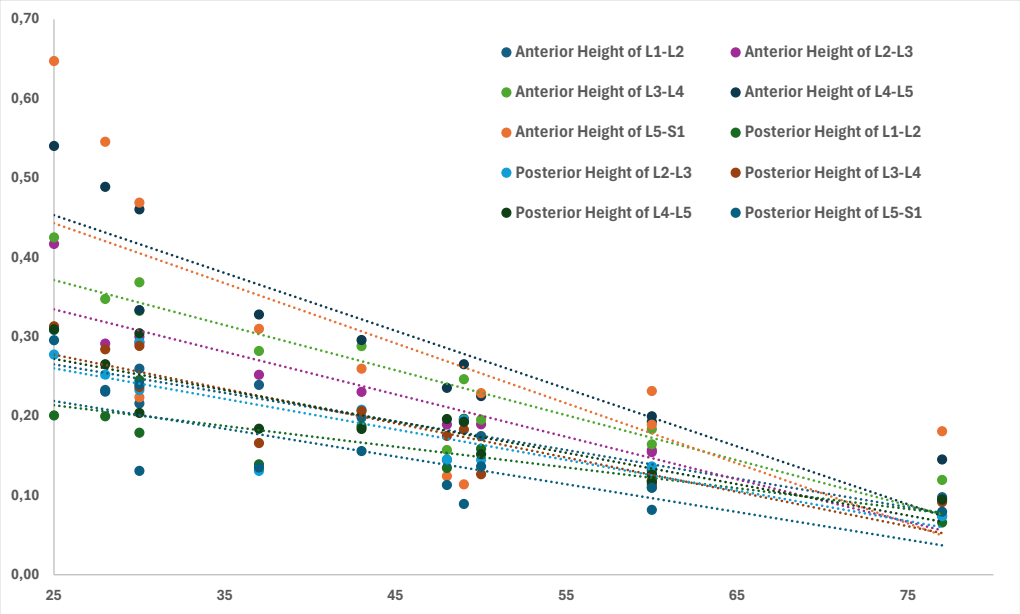

Figure 4.2 - Linear Regression between IVD height and Age in Group H.2.

Table 4.4 - Correlation between IVD height and Age in Group H.2.

Group H.2	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1-L2	-0,0036	0,8574	-0,0026	0,7051
L2-L3	-0,0054	0,8928	-0,0039	0,7634
L3-L4	-0,0057	0,8555	-0,0043	0,832
L4-L5	-0,0073	0,8206	-0,0039	0,8026
L5-S1	-0,0076	0,4908	-0,0035	0,6307

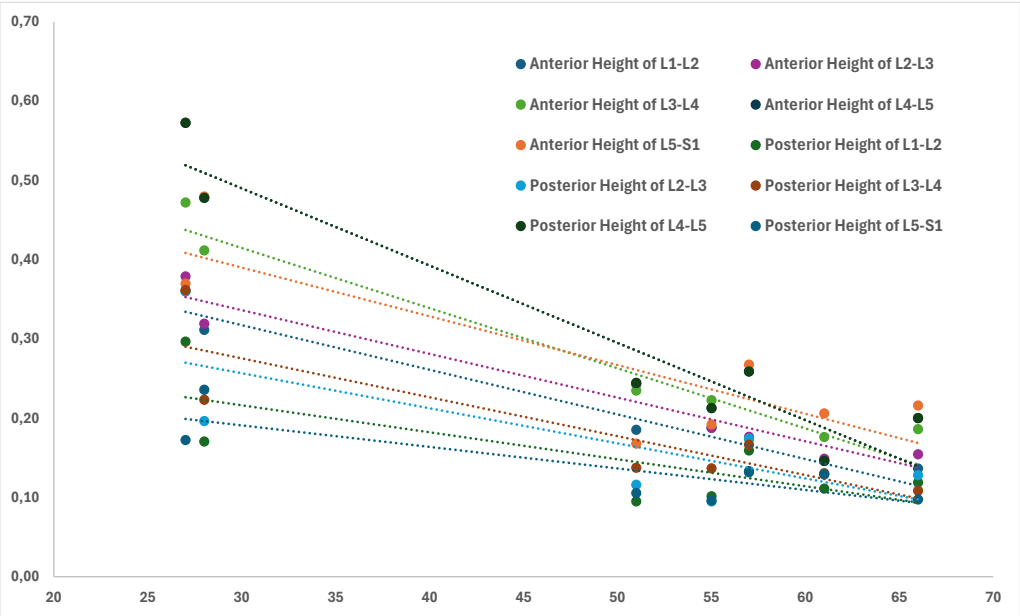

Figure 4.3 - Linear Regression between IVD height and Age in Group H.3.

Table 4.5 - Correlation between IVD height and Age in Group H.3.

Group H.3	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1-L2	-0,0056	0,9431	-0,0034	0,5686
L2-L3	-0,0055	0,9419	-0,0044	0,5861
L3-L4	-0,0076	0,9412	-0,0049	0,7596
L4-L5	-0,0097	0,9136	-0,0097	0,9136
L5-S1	-0,0061	0,7122	-0,0027	0,7048

Across all groups and lumbar levels examined, disc height decreases progressively with aging, although the rate and strength of this association varied not only between spinal levels, but also between patient groups defined by the location of LDH.

Group H.1 exhibited the smallest values of slope, particularly in the posterior measurements. These values indicate a comparatively modest reduction in IVD height with advancing age. In addition, Group H.1 demonstrated the lowest coefficients of determination (R^2), in some cases falling below 0.20, particularly in the posterior column. This suggests that age is a relatively weak predictor of disc height in this group, and that L4-L5 herniation in these individuals may be influenced by differences in disc morphology. Such structural variations could contribute to a more heterogeneous pattern of disc height change, which is reflected in the comparatively poor age-height correlations observed. This supports the notion that disc changes in this group may not follow the typical age-related degenerative pattern seen in the other groups.

In Group H.2 both anterior and posterior slopes were moderately more negative than those observed in Group H.1. This reflects a greater rate of disc height decline with aging. The R^2 values in this group were generally high at most lumbar levels, indicating a strong linear association between age and disc height. These results could indicate that L5-S1 herniation in this group is more closely associated with age degenerative processes. This pattern is particularly evident at the upper and mid-lumbar levels (L1-L4), where the relationship between age and disc height was most consistent.

Group H.3 demonstrated more abrupt slopes across nearly all levels, especially anteriorly. These values suggest the most rapid rate of disc height reduction with advancing age. Group H.3 also showed the consistently highest R^2 values, many above 0.90, indicating that age explains a substantial proportion of the variance in IVD height. The particularly strong correlations in the anterior column align with established biomechanical understanding that anterior disc structures may exhibit more predictable degenerative thinning over time [52].

These results indicate that the relationship between disc height and age is not uniform across patient groups. Patients from Group H.1 display the weakest age dependency, suggesting that factors beyond normal age-related degeneration may contribute to pathology in this group. On the contrary, Group H.3 show strong, predictable age-related reductions in disc height, consistent with more generalised degenerative disc disease. Group H.2 occupies an intermediate

position, demonstrating both age-related degeneration and potential mechanical or morphological contributions.

Overall, double-level herniation tends to occur in individuals who exhibit a more advanced pattern of disc degeneration, a profile more frequently observed in older age groups. In this sense, age predominantly worsens cases in which degeneration is the principal factor, while having a considerably smaller impact in cases where herniation is possibly caused principally by local biomechanical factors or anatomical characteristics.

4.2.3. IVD height vs BMI

In this sub-chapter the linear regression was performed to evaluate the relationship between IVD height and patient BMI (tables 4.6, 4.7 and 4.8).

Table 4.6 - Correlation between IVD height and BMI in Group H.1

Group H.1	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1-L2	0,0022	0,8993	-0,0018	0,4960
L2-L3	-0,0015	0,7127	-0,0022	0,6017
L3-L4	-0,0010	0,2197	-0,0010	0,5888
L4-L5	-0,0003	0,0127	-0,0012	0,5181
L5-S1	-0,0007	0,0804	-0,0004	0,0449

Table 4.7 - Correlation between IVD height and BMI in Group H.2.

Group H.2	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1-L2	-0,0015	0,6692	-0,0009	0,4791
L2-L3	-0,0020	0,8747	-0,0009	0,5182
L3-L4	-0,0018	0,6928	-0,0012	0,7203
L4-L5	-0,0025	0,8010	-0,0013	0,7008
L5-S1	-0,0023	0,3292	-0,0009	0,5010

Table 4.8 - Correlation between IVD height and BMI in Group H.3

Group H.3	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1-L2	-0,0009	0,7408	-0,0011	0,5932
L2-L3	-0,0007	0,7290	-0,0012	0,5166
L3-L4	-0,0010	0,8598	-0,0011	0,6783
L4-L5	-0,0010	0,4150	-0,0014	0,6105
L5-S1	-0,0022	0,7947	-0,0012	0,7344

In Group H.1, the strongest anterior correlations were found in L1-L2 and L2-L3, while the association was weak at the herniated level and at L5-S1. Posteriorly, the strongest relationships were seen at mid-lumbar levels L2-L3 and L3-L4, again weakening at L4-L5. These results may suggest that at the degenerated level, disc morphology is more strongly influenced by structural damage than by BMI, reducing the strength of BMI for disc height variability. In contrast, upper lumbar discs show a more predictable decline in height with increasing BMI, this could be because these levels remain relatively preserved and therefore have a more predictable mechanical response to body mass, whereas at the herniated level there's already structural changes in the disc, so the BMI effects are not as visible as in the upper levels.

Group H.2 demonstrated generally stronger and more uniform findings. Anteriorly, BMI had high association and explained a substantial proportion of IVD height variance, particularly at the middle of the lumbar spine. Even at the pathological level L5-S1, there was moderate value. Posterior measurements showed a similar pattern, with the highest R^2 at L3-L4 and L4-L5. Compared to Group H.1, the regression remained stronger near the affected level, which may reflect more uniform disc structural behaviour or a different degenerative timeline in L5-S1 herniation. The magnitude of the slopes was also steeper in some segments, particularly at L4-L5 and L5-S1 anteriorly and at L3-L4 posteriorly, although more mildly, indicating a more pronounced BMI-related decline in disc height at these levels.

Group H.3 displayed moderately strong relationships across most levels. Anterior R^2 values were highest at L3-L4 and L5-S1, suggesting substantial influence of BMI even at affected levels. Posterior R^2 values were also consistent, peaking at L5-S1 and L3-L4. Because this group presents with multi-level pathology, the persistence of a strong BMI-IVD relationship suggests that excess body mass may contribute to more extensive disc height loss, potentially accelerating degenerative changes across multiple lumbar levels.

These observations align with an existing study by Singh et al. 2024, where a large population-based MRI study found that higher BMI was significantly associated with the presence, extent, and severity of LDH. Overall, the data underlines the clinical importance of BMI as a modifiable risk factor in lumbar disc degeneration and suggests that strategies for weight management may be relevant not only for symptom control but for slowing structural spinal degeneration [53].

4.2.4. IVD Height vs Patient Height

The analysis between IVD height and patient height is done in this sub-section and it's divided between the three groups, as it's represented in tables 4.9, 4.10 and 4.11.

Table 4.9 - Correlation between IVD height and Patient Height in Group H.1.

Group H.1	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1-L2	0,0002	0,1141	-0,0005	0,2479
L2-L3	0,0000	0,0009	-0,0006	0,2734
L3-L4	0,0001	0,0104	-0,0003	0,3569
L4-L5	-0,0001	0,0085	0,0000	0,0110
L5-S1	-0,0004	0,0673	-0,0002	0,0367

Table 4.10 - Correlation between IVD height and Patient Height in Group H.2.

Group H.2	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1-L2	-0,0004	0,2485	-0,0005	0,2204
L2-L3	-0,0007	0,4286	-0,0002	0,0620
L3-L4	0,0000	0,0002	-0,0001	0,0349
L4-L5	-0,0002	0,0393	-0,0002	0,0751
L5-S1	0,0001	0,0019	-0,0002	0,0585

Table 4.11 - Correlation between IVD height and Patient Height in Group H.3.

Group H.3	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1-L2	0,0001	0,0484	-0,0006	0,2510
L2-L3	-0,0003	0,2988	-0,0005	0,1259
L3-L4	0,0002	0,2003	-0,0003	0,0841
L4-L5	0,0002	0,0117	0,0002	0,0182
L5-S1	-0,0015	0,7139	-0,0001	0,8219

In Group H.1, slopes were extremely small, ranging from slightly positive to slightly negative in both anterior and posterior measurements. This indicates that disc height does not systematically increase or decrease with patient height. The R^2 values were uniformly low (mostly below 0.30 and often below 0.10), showing that only a small proportion of variation in disc height can be attributed to patient stature. These results suggest that, in this group, disc height variability is largely independent of overall body height. Group H.2 showed a similar pattern. Although a few levels presented slightly higher R^2 values, most values remained very low, particularly in the posterior measurements. The slopes again approached zero, indicating no meaningful linear relationship between height and IVD dimensions. Overall, these results show that patient height

has minimal influence on disc height patterns along the lumbar spine. The consistently small slopes and low R^2 values demonstrate that height does not predict these differences between segments. Thus, the relative distribution of disc height across lumbar levels appears more closely related with local biomechanical and degenerative patterns than with overall body size.

Group H.3 displayed the same general trend of very low slopes and weak correlations. One exception was L5-S1, where R^2 values were higher (0.71 anterior, 0.82 posterior), suggesting that at this level patient height may account for some variability in disc height. However, this pattern was not observed at other levels, and the slopes even at L5-S1 remained small. Thus, the biological effect of height is likely limited.

These results are consistent with the observations reported by Çetin et al. 2023, who found that at the L5-S1 level, herniated discs did not differ significantly in height from non-herniated discs, whereas at L4-L5, herniated discs were significantly smaller. This pattern indicates that disc height alterations associated with herniation are level-dependent and not simply a consequence of patient stature. Instead, it reinforces the notion that localised degeneration, biomechanical environment, and disc-specific structural characteristics play a more decisive role in determining disc height than body height alone [35].

4.3. Characterization of Vertebrae Height

Quantifying vertebral height differences provides structural information that can be useful for computational modelling approaches such as FEA, as segmental geometry and dimensional changes influence load distribution and deformation behaviour. In this section, the height of the vertebrae across the lumbar segments is analysed. The average values of the vertebrae height are in Appendix B.

4.3.1. Vertebrae Height Difference across Levels

To study this parameter, the equation 3.1 was applied to the mean values of the 3 measurements. The results are presented in Table 4.12.

Table 4.12 - Average difference in levels of vertebrae height in patients.

	L1-L2		L2-L3		L3-L4		L4-L5		L5-S1	
	A	**P**	**A**	**P**	**A**	**P**	**A**	**P**	**A**	**P**
Group H.1	6%	20%	6%	0%	-5%	-8%	3%	-10%	-23%	12%
Group H.2	6%	4%	3%	-1%	-1%	-4%	0%	-8%	17%	5%
Group H.3	4%	2%	5%	-1%	-3%	-3%	5%	-13%	-14%	10%

A - Anterior; P - Posterior.

In Group H.1, the largest differences in vertebral height occurred in the lower lumbar levels, especially between L5 and S1. However, this transition naturally presents greater height variation because L5 and S1 have distinct anatomical morphologies. Therefore, the size of the L5-S1 difference reflects the anatomical characteristics of the lower spine rather than degeneration. Differences in the upper levels were comparatively smaller. In Group H.2, the most

pronounced difference also appeared at the L5-S1 transition, with smaller variations in the remaining levels. This pattern mainly reflects the anatomical difference between these two vertebrae. In Group H.3, larger differences were again found at the lower lumbar transitions, while upper lumbar transitions showed reduced variation.

According to the literature, healthy vertebral height typically increases progressively from L1 to S1 [54]. In the present study, the results do not consistently demonstrate this progressive pattern across all groups. The observed differences across levels, especially at L5-S1, primarily reflect the natural morphological variation of vertebrae rather than degenerative change.

4.3.2. Vertebrae Height vs Age

Analysis of vertebrae height and the age of each participant in the database was made separately in the three groups of study (Tables 4.13, 4.14 and 4.15).

Table 4.13 - Correlation between vertebrae height and age in Group H.1.

Group H.1	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1	-0,0088	0,9381	-0,0112	0,8474
L2	-0,0108	0,9102	-0,0116	0,8547
L3	-0,0107	0,9303	-0,0128	0,8204
L4	-0,0098	0,8977	-0,0108	0,9077
L5	-0,0099	0,9077	-0,0112	0,9112
S1	-0,0127	0,9547	-0,0099	0,9052

Table 4.14 - Correlation between vertebrae height and age in Group H.2.

Group H.2	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1	-0,0124	0,884	-0,0136	0,8663
L2	-0,0133	0,9307	-0,0146	0,8474
L3	-0,014	0,9024	-0,0145	0,8693
L4	-0,0144	0,8496	-0,0140	0,8503
L5	-0,0145	0,8129	-0,0117	0,873
S1	-0,0155	0,8696	-0,0128	0,8097

Table 4.15 - Correlation between vertebrae height and age in Group H.3.

Group H.3	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1	-0,0164	0,9776	-0,0198	0,9715
L2	-0,0175	0,9733	-0,0182	0,9737
L3	-0,0186	0,9802	-0,0195	0,9513
L4	-0,0175	0,9686	-0,0185	0,9686
L5	-0,0178	0,9642	-0,0172	0,9519
S1	-0,02	0,9204	-0,0138	0,987

Group H.1 showed a gradual decrease in vertebral height with age across all lumbar levels. The slopes indicate that vertebral height decreases gradually over time. High R^2 values demonstrate that age accounts for most of the variation in vertebral height in this group. These results suggest that in patients with isolated L4-L5 herniation, vertebral body changes are predictable and largely due to age, but the magnitude of structural loss is relatively small, possibly reflecting early or mild degeneration. Group H.2 exhibited a steeper decline in vertebral height compared with Group H.1, with consistently high R^2 values. Although vertebral height showed a strong association with age, vertebral bodies typically undergo only minor age-related changes, and the high R^2 values likely reflect small but uniform remodelling rather than meaningful degeneration. In contrast, disc height varies due to several degenerative mechanisms, which explains its weaker correlation with age. Group H.3 demonstrated the steepest slopes out of the three groups, reflecting the most pronounced vertebral height loss with age. The very high R^2 values (>0.95) indicate that these changes are highly predictable. The steep slopes can potentially reflect age-related reductions in vertebral height, not an effect of the herniation, since disc pathology does not alter vertebral bone structure.

Although the statistical strength of the associations was high, the actual reductions in vertebral height were mild and may have limited biomechanical relevance. The strong correlations mainly reflect how stable and consistent vertebral height is with aging, whereas disc height is influenced by a extensive range of degenerative processes, such as BMI.

4.3.3. Vertebrae Height vs BMI

Analysis of vertebrae height and the BMI of each participant in the database was made separately in the three groups of study, has seen in tables 4.16, 4.17 and 4.18.

Table 4.16 - Correlation between vertebrae height and BMI in Group H.1.

Group H.1	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1	-0,0231	0,8269	-0,0283	0,8509
L2	-0,0306	0,8146	-0,0332	0,8991
L3	-0,0287	0,9149	-0,0308	0,7482
L4	-0,0269	0,8342	-0,0264	0,8615
L5	-0,0241	0,8386	-0,0281	0,8135
S1	-0,0348	0,8651	-0,0287	0,6882

Table 4.17 - Correlation between vertebrae height and BMI in Group H.2.

Group H.2	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1	-0,0346	0,8112	-0,0342	0,7395
L2	-0,0398	0,9494	-0,0359	0,7300
L3	-0,0365	0,8461	-0,0379	0,7808
L4	-0,0355	0,7715	-0,0365	0,7430
L5	-0,0345	0,7081	-0,0346	0,7607
S1	-0,0403	0,8374	-0,0333	0,6889

Table 4.18 - Correlation between vertebrae height and BMI in Group H.3.

Group H.3	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1	-0,024	0,9559	-0,0181	0,7168
L2	-0,0202	0,9766	-0,0186	0,7019
L3	-0,0182	0,9470	-0,0199	0,6474
L4	-0,0253	0,9226	-0,0158	0,6141
L5	-0,0174	0,6972	-0,0145	0,6261
S1	-0,0349	0,8499	-0,0266	0,9116

Linear regression analysis of vertebral height versus BMI revealed negative slopes across all lumbar levels and groups, indicating that higher BMI was generally associated with slightly reduced vertebral heights. The results contrast with the analysis of IVD height versus BMI, suggesting that BMI relates more to vertebral height than to disc height, which in this case appeared to be affected mainly by how degenerated each disc was.

In Group H.1, the relationship was relatively uniform across the lumbar spine, with R^2 values generally above 0.8, particularly at anterior height of L3 that was the highest value. Group H.2 displayed slightly steeper anterior slopes at L2 and S1, although R^2 values were more variable, suggesting that factors other than BMI may influence vertebral height, such as age. Group H.3 showed the steepest relation at S1, with posterior R^2 also high at this level, indicating that vertebral height reductions were most pronounced in the lowest lumbar segment when multiple herniations were present.

These results indicate a moderate negative association between BMI and vertebral height across lumbar levels (the higher the BMI is, vertebral height tends to decrease). Given the rigid nature of vertebrae, changes in height likely reflect structural alterations, such as degenerative changes. The association between BMI and vertebral height varied across lumbar levels, with some vertebrae showing stronger negative correlations than others, but no consistent pattern directly related to the location of herniated discs was observed.

4.3.4. Vertebrae Height vs Patient Height

In this sub-chapter the analysis of vertebrae height versus the patient height was made using linear regression to evaluate the relationship between the two variables. Tables 4.19, 4.20, and 4.21 present the trend line of each linear regression for the patients in each group, divided by spinal level and by height (anterior or posterior).

Table 4.19 - Correlation between vertebrae height and patient height in Group H.1.

Group H.1	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1	-0,0007	0,3326	-0,0003	0,1025
L2	-0,0003	0,1087	-0,0002	0,0534
L3	-0,0009	0,7759	0,0004	0,1594
L4	-0,0004	0,3254	-0,0002	0,0752
L5	-0,0007	0,3529	0,0000	0,0009
S1	-0,0006	0,3426	-0,0005	0,096

Table 4.20 - Correlation between vertebrae height and patient height in Group H.2.

Group H.2	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1	-0,0001	0,0190	0,0002	0,0620
L2	-0,0006	0,3479	0,0001	0,0128
L3	-0,0004	0,1360	-0,0001	0,0182
L4	-0,0002	0,0186	0,0000	0,0007
L5	0,0000	0,0014	0,0000	0,0034
S1	0,0002	0,0325	0,0006	0,1937

Table 4.21 - Correlation between vertebrae height and patient height in Group H.3.

Group H.3	Anterior		Posterior	
	Slope	R^2	Slope	R^2
L1	-0,0005	0,4706	0,0006	0,2515
L2	0,0000	0,0477	0,0001	0,0057
L3	0,0000	0,0081	0,0005	0,0788
L4	-0,0002	0,0467	0,0005	0,1025
L5	0,0002	0,0253	0,0007	0,3703
S1	-0,0002	0,0233	-0,0011	0,7105

In Group H.1, various anterior vertebral measurements demonstrated comparatively to posterior, higher correlations with patient height. Notably, the anterior height of L3 showed the strongest association, followed by moderate relationships at L5 and S1. In contrast, posterior vertebral heights exhibited weaker associations, with R^2 values typically below 0.20. Despite these variations, the slopes across all vertebrae remained small in magnitude, indicating minimal change in vertebral height relative to patient height. In Group H.2 the relationships between patient height and vertebrae height changes from upper to lower spine were consistently weak. R^2 values for both anterior and posterior measurements were generally below 0.05, with few

exceptions. These results suggest that, in this group, vertebral height variability was largely independent of patient height, and that no vertebral level demonstrated a substantial linear association. In Group H.3 the overall pattern was similar to that observed in Groups 1 and 2, with weak correlations across most vertebral levels. However, posterior S1 height demonstrated a comparatively strong association with patient height, representing the strongest relationship identified in this group. Additional moderate correlations were observed at anterior and at posterior L5. The remaining vertebral levels exhibited low R^2 values, indicating limited relation of vertebral height on overall body height.

The results indicate that patient height is possibly not a strong predictor of lumbar vertebral patient height changes in any of the groups studied. Although isolated vertebral levels displayed moderate or strong correlations, these were exceptions. The results across the dataset suggest that vertebral height is influenced predominantly by factors other than patient height, such as degenerative changes, localized biomechanical loading, and anatomical variability.

4.4. Characterization of Lumbar Lordosis Angle (LLA)

The LLA represents a critical anatomical parameter in the assessment of sagittal alignment and overall spinal curvature. Alterations in this angle can indicate postural compensation or pathological changes associated with LDH. The following section examines the variation of the LLA across vertebral levels and explores its relationship with patient age and other parameters. The average values of the LLA are in Appendix B.

4.4.1. Lumbar Lordosis Angle by Level

To analyse this measurement, the mean LLA was calculated for each group. In a study of 324 asymptomatic adults with a mean age of 55 years, a mean LLA of 50.3° was reported when measured in the standing position using X-ray [55].

Group H.1 had a mean LLA of 42.40°; Group H.2 had a mean LLA of 49.74°; and Group H.3, had a mean LLA of 42.13°. These results indicate that patients with a disc herniation involving at least the L4-L5 level tend to present with a reduced lumbar lordosis angle. The values for the LLA from each patient are presented in appendix B.

This observation aligns with the results of Zhou et al. 2024, where the relationship between LDH and spinal alignment was investigated. Their study demonstrated that herniation at L4-L5 is associated not only with a decrease in disc height and disc angle but also with a reduction in overall lumbar lordosis and sacral slope. The authors suggest that these changes reflect biomechanical adaptations of the lumbar spine to disc degeneration and herniation, which may alter load distribution and spinal curvature. The study also indicated that L5-S1 herniation alone does not significantly affect lumbar lordosis, supporting the idea that the L4-L5 level plays a more critical role in maintaining normal sagittal alignment [56].

4.4.2. Lumbar Lordosis Angle by Age

The relationship between the LLA and age was also examined across the three patient groups with varying disc herniation levels. The regression equations and R^2 for each group are as follows in Table 4.22.

Table 4.22 - Linear Regression between LLA and Age.

	Group H.1	Group H.2	Group H.3
Slope	-0,0067	-0,0237	-0,0124
R^2	0,0928	0,7834	0,6274

In Group H.1 with a mean age of 53 years, showed a small association between age and LLA, suggesting minimal age-related change in lumbar curvature for patients with isolated L4-L5 herniation. In contrast, Groups 2 and 3 exhibited moderate negative associations, indicating that LLA tends to decrease with age in patients with L5-S1 or herniations at the two lowest levels. Although Group H.1 had a slightly higher mean age than Groups 2 and 3, the effect of age on LLA was minimal, suggesting that factors such as herniation location, individual anatomy, or disc degeneration could potentially influence spinal angle independently of chronological age.

These findings are broadly consistent with the observations of Skaf et al. 2011, who reported that LLA tends to decrease with increasing age, and that disc herniations occur at higher lumbar levels in older individuals. Their observations indicated that younger patients generally exhibit higher LLA and experience disc herniations at lower lumbar levels, whereas older patients have reduced lumbar lordosis and tend to develop herniations at higher levels. They attributed this shift to age-related changes in spinal biomechanics, including alterations in load distribution and sagittal alignment [16]. Our data show that patients with herniations at lower lumbar levels (Groups H.2 and H.3) tend to experience a more pronounced reduction in lumbar lordosis with age, while Group H.1 appears less influenced by age-related curvature changes. Differences in sample characteristics, measurement techniques, or the presence of chronic adaptations related to pain, muscular imbalance, or disc height loss may have contributed to some of the distinct patterns observed in this study.

4.4.3. Lumbar Lordosis Angle by BMI

The relationship between the LLA and age was also examined across the three patient groups with varying disc herniation levels. The regression equations and R^2 for each group are as follows in Table 4.23.

Table 4.23 - Linear Regression between LLA and BMI

	Group H.1	Group H.2	Group H.3
Slope	-0,0347	-0,0779	-0,0510
R^2	0,1380	0,7066	0,2921

Group H.1 showed a small negative slope and low R^2, suggesting only a very weak relationship between BMI and lumbar curvature in these patients. Group H.2 exhibited a vertical negative slope with a relatively high R^2, indicating a moderate association in which higher BMI may contribute to a reduction in lumbar lordosis in this subgroup. Group H.3 had an intermediate slope and low R^2, reflecting a moderate negative association with considerable variability.

These results suggest that BMI may influence lumbar lordosis to a limited extent, with the effect being more pronounced in Group H.2. In contrast, groups H.1 and H.3 show weaker and more variable associations. While higher BMI could potentially contribute to mechanical loading on the lumbar spine, the variability and generally low explanatory power indicate that other factors, such as age, herniation location, and degenerative changes, are likely more influential determinants of lumbar curvature.

4.5. Characterization of Intervertebral Angle

The intervertebral angle quantifies the relative inclination between adjacent vertebral endplates, serving as a biomechanical indicator of segmental mobility and spinal curvature. Changes in this angle may appear from degenerative disc processes, mechanical overload, or compensatory alignment. In this section, both sagittal and coronal intervertebral angles are analysed to provide an interpretation of lumbar segmental changing aspects. The average values of the intervertebral angle are in Appendix B.

4.5.1. Intervertebral Angle in Sagittal Plane

In this sub-chapter, the analysis of intervertebral angles in sagittal plane is represented in table 4.24. In this study the angle measurements showed a high percentage deviation between repetitions (10% to 22%). Therefore, despite being theoretically more sensitive, the angular measurements were one of the less reliable, which limits the strength of conclusions based on angle changes.

Table 4.24 - Average difference of intervertebral angle between levels in sagittal plane.

	L1-L2 to L2-L3	L2-L3 to L3-L4	L3-L4 to L4-L5	L4-L5 to L5-S1
Group H.1	103%	84%	9%	225%
Group H.2	193%	121%	60%	41%
Group H.3	-20%	129%	145%	32%

In healthy subjects, the intervertebral angle progressively increases from the upper to the lower lumbar levels, with L4-L5 and L5-S1 typically presenting the largest values [55]. By analysing the results in Table 4.24, an increase in the intervertebral angle from L1 to S1 was generally observed across all groups, although in Group H.1 the increase from L3-L4 to L4-L5 was only 9% and in Group H.2 the minimum was 41% and in Group H.3 was of 32%. This smaller increase could be due to the localized biomechanical disruption caused by the herniation. Similarly, in Group H.2, the angular increase between L4-L5 and L5-S1 was reduced to 40%,

corresponding to the affected level. These patients exhibited a 60% increase from L3-L4 to L4-L5, suggesting that the superior adjacent disc may experience compensatory biomechanical loading. In cases with herniations at both L4-L5 and L5-S1, only a 32% increase was observed at the herniated levels, while the adjacent segments showed no substantial differences, except for the upper lumbar levels (L1-L3), where a slight reduction in intervertebral angle was noted.

Overall, although intervertebral angles tend to increase from upper to lower levels, this progression is consistently interrupted at herniated segments due to reduced disc height and loss of disc material.

4.5.1.1. Intervertebral Angle vs Age

The intervertebral angle was measured for all patients, and the analysis was divided according to each group (Table 4.25).

Table 4.25 - Correlation between intervertebral angle and age.

	Group H.1		Group H.2		Group H.3	
	Slope	**R^2**	**Slope**	**R^2**	**Slope**	**R^2**
L1-L2	-0,0017	0,1054	-0,0027	0,3367	-0,0049	0,664
L2-L3	-0,0031	0,3016	-0,0026	0,3633	-0,0025	0,2328
L3-L4	-0,0036	0,6020	-0,0036	0,4665	-0,0059	0,5920
L4-L5	0,0002	0,0028	-0,0075	0,7084	-0,0051	0,7449
L5-S1	-0,0036	0,2291	-0,0123	0,5967	-0,0076	0,6574

Overall, the sagittal-plane analysis showed a tendency for intervertebral angles to decrease with age at most lumbar levels, although this trend was not uniform across all segments. This pattern is broadly consistent with the disc-height findings, since reductions in disc height can contribute to a loss of disc angle; however, angular behaviour proved more variable.

In Group H.1, age showed generally weak associations, with most R^2 values at or below 0.30, except at L3-L4. At L4-L5, the slope was near zero, indicating no meaningful age effect at the herniated level, which mirrors the weaker age-height relationships found in this group.

In Group H.2, all lumbar segments showed negative slopes, but the associations were moderate. The highest R^2 values were found at L4-L5 and L5-S1, suggesting that age has a clearer but still moderate influence on angulation near the herniated segment and its adjacent level.

In Group H.3, age-related reductions in sagittal angulation were observed across multiple levels, but the strength of association was again moderate.

Overall, age-related reductions in sagittal angulation follow the same general direction as disc-height changes, weakest in Group H.1, moderate in Group H.2 and H.3, however angular measurements are less sensitive and more variable, making these associations less consistent than those observed for disc height.

4.5.1.2. Intervertebral Angle vs BMI

In this sub-chapter, the analysis between intervertebral angles and BMI in sagittal plane is represented in table 4.26.

Table 4.26 - Correlation between intervertebral angle and BMI

	Group H.1		Group H.2		Group H.3	
	Slope	**R^2**	**Slope**	**R^2**	**Slope**	**R^2**
L1-L2	-0,0077	0,1769	-0,0116	0,3762	-0,0113	0,2965
L2-L3	-0,0088	0,2765	-0,0137	0,6381	-0,0035	0,0281
L3-L4	-0,0095	0,2481	-0,0098	0,4083	-0,0116	0,1901
L4-L5	-0,0217	0,5832	0,0038	0,0440	-0,0139	0,3897
L5-S1	-0,0431	0,5722	-0,0007	0,0039	-0,0253	0,5861

The evaluation of intervertebral angle in the sagittal plane demonstrated a general tendency for angular values to decrease with increasing BMI across most lumbar levels, although the strength and direction of these associations varied between groups and segments.

In Group H.1, all lumbar levels demonstrated decreasing intervertebral angles with increasing BMI. The most notable relationships were observed at L4-L5 and L5-S1, suggesting that higher BMI may have a more evident impact on the lower lumbar spine in this group. In Group H.2, moderate associations were found at L2-L3 and L3-L4, indicating that BMI may influence more superior levels in this group. However, at L4–L5 the slope was slightly positive and the association weak, meaning that BMI did not meaningfully reduce the angle at the herniated level in this group. At L5–S1, the effect of BMI was insignificant. In Group H.3, decreasing intervertebral angles were again present at most levels, although the strength of association was generally moderate. The clearest relationships were at L5-S1 and, L4-L5, suggesting that BMI may have a greater impact in the lower lumbar region when multiple levels are involved.

The data suggests that higher BMI is generally associated with reduced sagittal intervertebral angulation, particularly at L4-L5 and L5-S1. However, this relationship is not uniform across groups, and the influence of BMI appears more variable and segment-dependent than that observed for age.

4.5.2. Intervertebral Angle in Coronal Plane

In the coronal plane (Table 4.27), a general tendency for angular increase from the upper to the lower lumbar levels was observed across most groups, with the exception of patients with isolated L5-S1 herniation, who showed a reduction in angle between L1-L2 and L2-L3. Patients with two-level herniations also demonstrated smaller increases between L2-L3 and L3-L4, as well as a reduced angular change from L4-L5 to L5-S1. However, the coronal angle measurements exhibited substantial variability, with percentage deviations ranging from 10% to 43% across the three repetitions.

Table 4.27 - Average difference in intervertebral angle between levels in coronal plane.

	L1-L2 to L2-L3	L2-L3 to L3-L4	L3-L4 to L4-L5	L4-L5 to L5-S1
Group H.1	27%	52%	56%	73%
Group H.2	-24%	59%	47%	86%
Group H.3	19%	18%	74%	24%

In the coronal plane, the three groups displayed distinct patterns of angular behaviour. Group H.1 showed the largest and most consistent increases in intervertebral angle between consecutive lumbar levels, particularly towards the lower lumbar spine. In contrast, Groups H.2 and H.3 exhibited smaller or more irregular angular variations, especially at the herniated levels, suggesting that coronal-plane changes in these patients are more localized and influenced by segmental pathology.

Overall, the results indicate that angular behaviour in the coronal plane differs between groups, with the most uniform progression observed in patients with isolated L4-L5 herniation, while those with L5-S1 or two-level herniations display more variable patterns.

4.5.2.1. Intervertebral Angle vs Age

The correlation between intervertebral angles and age in coronal plane is represented in table 4.28.

Table 4.28 - Correlation between intervertebral angle and age.

	Group H.1		Group H.2		Group H.3	
	Slope	**R^2**	**Slope**	**R^2**	**Slope**	**R^2**
L1-L2	-0,0004	0,2474	-0,0017	0,5324	-0,0005	0,2286
L2-L3	0,0004	0,1268	-0,0014	0,4836	0,0000	0,0027
L3-L4	-0,0005	0,1825	-0,0009	0,5216	-0,0010	0,2776
L4-L5	0,0003	0,0334	-0,0008	0,2658	-0,0006	0,1313
L5-S1	-0,0007	0,3702	-0,0005	0,1046	-0,0002	0,0320

The results revealed substantially weaker and less consistent associations with age when compared with the sagittal plane. Angular variation across the lumbar spine in this plane appeared to be only minimally affected by aging.

In Group H.1, most levels demonstrated weak associations, with only L5-S1 showing a modest relationship. In Group H.2, moderate age-related associations were found at L1-L2 and L3-L4, although slopes were small, indicating relatively minor angular change. In Group H.3, age explained very little variability at any segment, and slopes remained close to zero, suggesting small coronal angular change attributable to age.

Overall, the results indicate that age has only a limited effect on coronal intervertebral angle, and that changes in this plane are likely more affected by measurement variability than by age-related structural differences.

4.5.2.2. Intervertebral Angle vs BMI

The correlation between intervertebral angles and BMI in coronal plane is represented in table 4.29.

Table 4.29 - Correlation between intervertebral angle and BMI.

	Group H.1		Group H.2		Group H.3	
	Slope	**R^2**	**Slope**	**R^2**	**Slope**	**R^2**
L1-L2	-0,0063	0,4716	-0,0004	0,0312	0,0004	0,0047
L2-L3	-0,0047	0,3921	-0,0008	0,0269	0,0015	0,0421
L3-L4	-0,0030	0,3244	-0,0011	0,1123	0,0022	0,1435
L4-L5	-0,0018	0,0875	-0,0007	0,0104	0,0010	0,0113
L5-S1	-0,0010	0,0166	-0,0007	0,0646	0,0011	0,0395

In the coronal plane, the association between intervertebral angle and BMI was considerably weaker compared to the sagittal plane.

In Group H.1, intervertebral angles decreased slightly as BMI increased, with the highest explanatory values observed at L1-L2 and L2-L3. In Group H.2, BMI showed minimal influence, with all R^2 values remaining low, indicating very limited explanatory contribution. In Group H.3, slopes were near zero and R^2 values were consistently low, suggesting no meaningful association between BMI and intervertebral angle in the coronal plane.

Overall, these data indicate that BMI does not significantly affect intervertebral angle in the coronal plane. Unlike the sagittal findings, the frontal alignment of the lumbar spine appears to be relatively independent of BMI, possibly reflecting a greater influence of other factors, such as asymmetric degeneration, mild scoliosis, anatomical variation, or measurement error.

4.6. Characterization of Pfirrmann Classification

The Pfirrmann classification is used to assess the degree of intervertebral disc degeneration, comprising five grades in total. Grade I indicates a healthy disc, whereas Grade V corresponds to a completely degenerated disc.

According to the results presented in Figure 4.4, from L1-L2 to L5-S1, the Pfirrmann grades progressively increase from the lower grades of degeneration (ranging from healthy to moderate degeneration, Grades I and II) to Grade V, indicating complete degeneration in the last level of the spine.

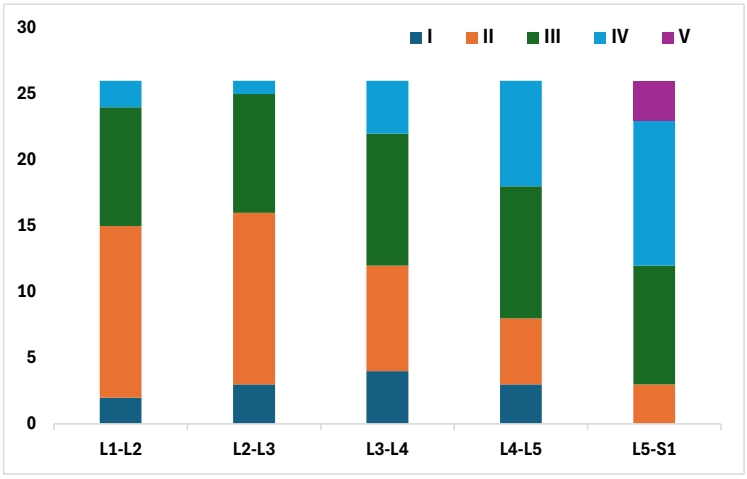

Figure 4.4 - Distribution of Pfirrmann classification by level of all the patients.

4.6.1. Pfirrmann Classification by Age

The relationship between Pfirrmann classification and age is explored in Figure 4.5.

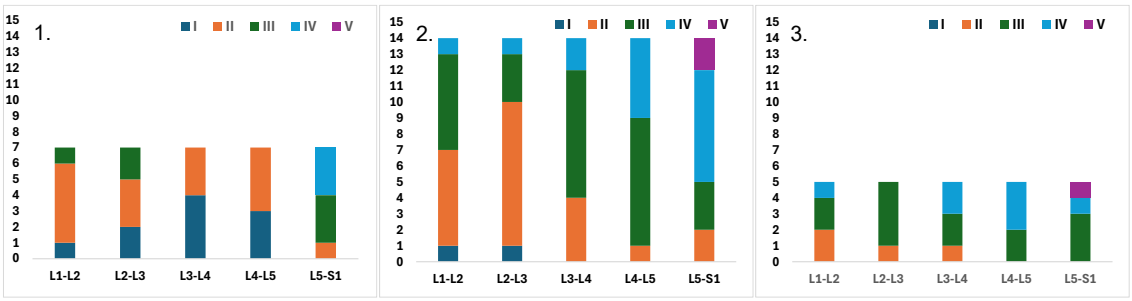

Figure 4.5 - Classification of Pfirrmann divided by age; Group A.1 - Patients with between 21 and 40 years old; Group A.2 - Patients with between 41 and 60 years old; Group A.3 - Patients with between 61 and 80 years old.

Patients in Group A.1 (21-40 years) presented the least extensive disc degeneration among the three groups. Degenerative changes were predominantly limited to the lower spine, and no discs in this age range reached the most advanced Pfirrmann grade (V). In nearly all cases, degeneration was confined to the disc directly affected by the herniation, most commonly at L5-S1, with the classification ranging from early-stage to moderate severity. Importantly, adjacent discs showed minimal to no degenerative alterations, indicating that in younger individuals the degenerative process tends to remain highly localized, even when a herniation is present. This pattern suggests a biomechanical rather than a generalized degenerative origin, since upper lumbar levels in this age group were consistently preserved, and multilevel degenerative changes were not identified.

In Group A.2 (41-60 years), disc degeneration became more frequent and more widely distributed across the lumbar spine. At the level of herniation, whether L4-L5, L5-S1, or both, degeneration was consistently moderate to severe, as expected in symptomatic patients within

this age range. However, unlike in Group A.1, adjacent discs often showed clear signs of additional degeneration. For example, patients with L5-S1 herniation commonly presented mild to moderate degeneration at L4-L5, and individuals with isolated L4-L5 herniation displayed varying degrees of involvement at L3-L4 or L5-S1. Although the degeneration of adjacent discs was not the same in every patient, the overall pattern shows that degeneration in this age group is no longer limited to one disc but often spreads to the adjacent levels. Some patients also had herniations at two consecutive levels, and in these cases, degeneration across several lumbar discs was more common, indicating a shift toward a broader and more widespread degenerative pattern.

Group A.3 (61-80 years) exhibited the most advanced and widespread degenerative patterns. Unlike the younger groups, patients older than 60 often showed degeneration at multiple lumbar segments, including the upper and mid-lumbar levels. Herniations in this group were frequently associated with moderate to severe disc degeneration not only at the affected level but also in the adjacent and even upper segments. The discs immediately superior to the herniated level rarely appeared healthy, and multilevel Pfirrmann grade III-IV classifications were common. This suggests that aging contributes to a general decline in disc structure, reducing the spine's capacity to compensate for biomechanical stress and making all levels more susceptible to degeneration. Even in patients with two herniated levels, degeneration was not limited to those segments but extended across the entire lumbar spine in a generalized pattern.

Taken together, these results reinforce a clear progression across age groups. Younger patients in Group A.1 display isolated, localized degeneration that is typically restricted to the level of the herniation, with preserved adjacent discs. Individuals in Group A.2 present a mixed pattern, where degeneration at the herniated level is accompanied by variable but increasingly frequent association of adjacent discs. By Group A.3, degeneration becomes widespread, with herniations occurring within a spine that is globally compromised. This suggests that both age and the presence of herniation influence the distribution of degenerative changes, but age appears to be a dominant factor in determining how extensive degeneration spreads beyond the symptomatic disc.

4.6.2. Pfirrmann Classification by BMI Classification

The present analysis demonstrates the relationship between increasing BMI and the degree disc degeneration assessed by the Pfirrmann classification (Figure 4.6).

Patients in Group B.1, classified as having normal weight, generally showed the lowest degree of disc degeneration across the lumbar spine. Degenerative changes tended to be mild and were most evident at L5-S1, which is consistent with the fact that most patients in this group presented herniation at this specific level. Although several individuals showed Grade II or Grade III changes at the upper levels, the pattern remained relatively localized, with small involvement of the adjacent discs. Even when a herniation was present, degeneration at adjacent segments

was limited or absent, suggesting that normal-weight individuals may maintain better disc integrity and that structural compromise often remains confined to the symptomatic level rather than spreading across the lumbar column.

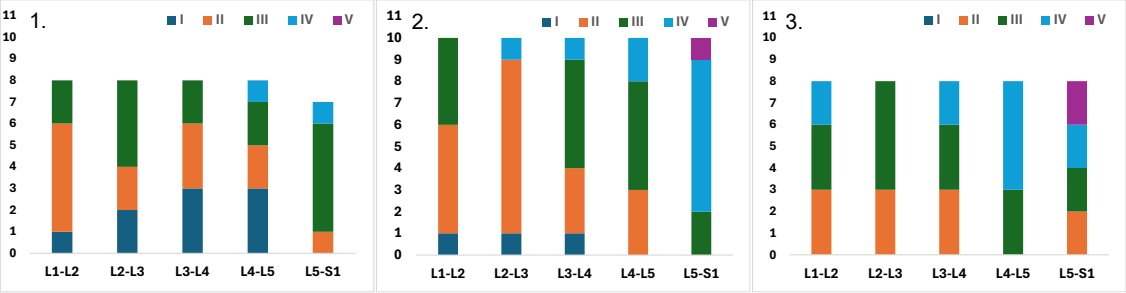

Figure 4.6 - Classification of Pfirrmann divided by BMI; Group B.1 - Patients with normal weight; Group B.2 - Patients overweight; Group B.3 - Patients with obesity grade I, II and III.

In Group B.2 (overweight patients), degeneration became more frequent and was distributed more broadly across the lumbar levels. A substantial proportion of these patients presented moderate to advanced degeneration, especially at L3-L4, L4-L5, and L5-S1. At the herniated level, degeneration commonly reached Grade III or IV, regardless of whether the herniation occurred at L4-L5, L5-S1, or both. Importantly, changes at the adjacent discs were more evident in this group. Patients with L5-S1 herniation often displayed degeneration at L4-L5, and those with L4-L5 herniation frequently showed involvement at L3-L4 and L5-S1. Although the extent of adjacent-level changes varied from patient to patient, the overall pattern indicated that being overweight was associated with a broader distribution of degenerative changes that no longer remained limited to the herniated disc alone. The presence of two-level herniations within this BMI category was accompanied by degeneration across multiple discs, particularly in the lower lumbar levels, providing further evidence that mechanical loading associated with increased body weight may accelerate the structural decline of adjacent discs.

Patients in Group B.3, which included individuals with obesity from Grade I to Grade III, displayed the most widespread and advanced degenerative changes among the three BMI groups. Many of these patients presented Grade III or IV degeneration across multiple lumbar levels, with some reaching Grade V at the herniated segment. Unlike the previous groups, degeneration of upper lumbar levels such as L1-L2 and L2-L3 was more frequent, reflecting a pattern of structural deterioration that extended beyond the lower lumbar region. Herniations in this group were rarely associated with degeneration confined to a single level; instead, adjacent discs almost always showed moderate to severe degeneration. Patients with two-level herniations tended to present degeneration affecting multiple lumbar segments, most prominently in the lower spine and in some cases reaching the mid-lumbar levels. This widespread pattern suggests that increased mechanical load and altered spinal biomechanics associated with obesity may

accelerate overall disc degeneration, leading to a more global rather than localized pattern of structural compromise.

Overall, the comparison across the three BMI categories demonstrates a clear trend where normal-weight individuals tend to exhibit localized and less advanced degeneration; overweight individuals show a broader and more variable pattern affecting multiple levels; and patients with obesity present the most extensive and severe degenerative changes throughout the lumbar spine. While the location of the herniation remains an important determinant of where degeneration first appears, BMI appears strongly associated with how far degeneration progresses beyond the herniated segment. The shift from a localized pattern in normal-weight individuals to a widespread and multilevel pattern in individuals with obesity suggests that body mass influences both the severity and distribution of lumbar disc degeneration.

4.6.3. Pfirrmann Classification by Level of LDH

When separating the patients by their groups, the results differ group by group (Figures 4.7, 4.8 and 4.9). Group H.1 includes patients with herniation at L4-L5, Group H.2 contains patients with herniation at L5-S1, and Group H.3 consists of patients presenting herniations at both lumbar levels.

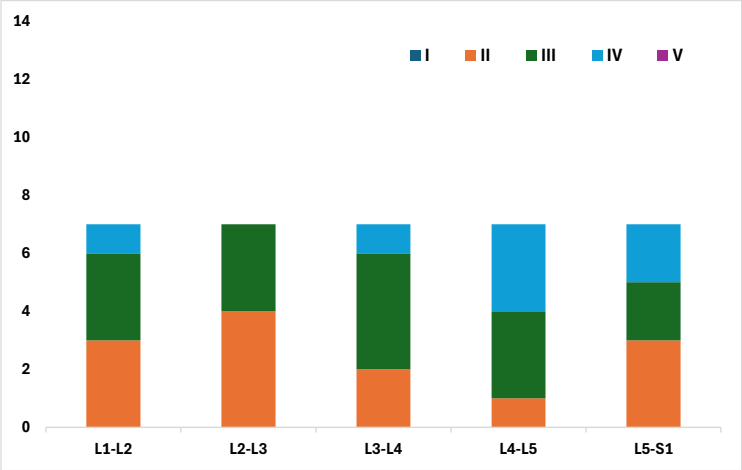

Figure 4.7 - Pfirrmann Classification of Group H.1.

According to the results, patients in Group H.1 exhibit variations throughout the lumbar spine from the upper to the lower levels, with the highest Pfirrmann grade (Grade IV) observed at the herniated level. The superior and inferior adjacent discs show moderate degeneration, which, according to the classification, already indicates a reduction in disc height.

When analysing Figure 4.8, it can be observed that the most degenerated level corresponds to the herniated segment. However, there is also a gradual increase in degeneration from the upper (L1-L2) to the lower (L5-S1) lumbar levels, evidenced by a decrease in Grade I (healthy) discs and a rise in Grade III (moderate degeneration) discs.

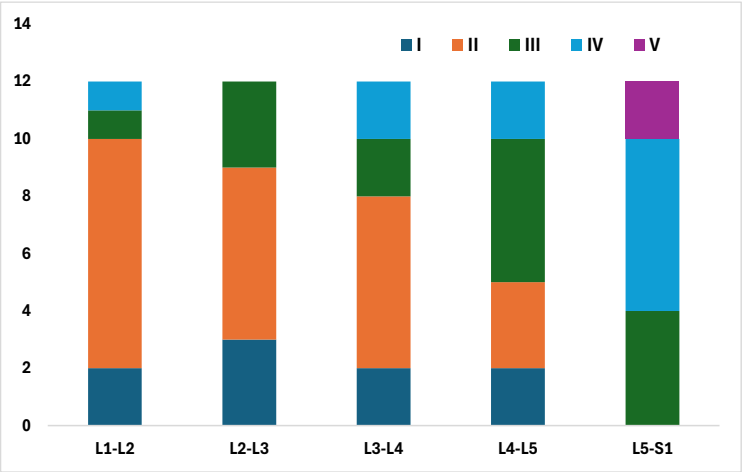

Figure 4.8 - Pfirrmann Classification of Group H.2.

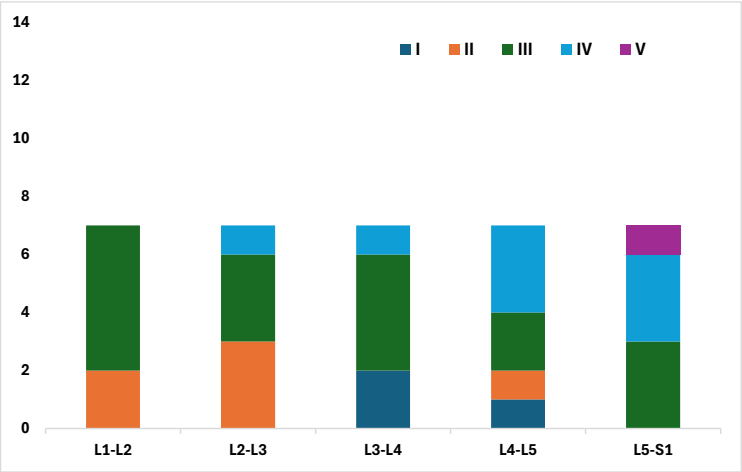

Figure 4.9 - Pfirrmann Classification of Group H.3.

Figure 4.9 results correspond to Group H.3, which includes patients with disc herniations at the two lowest lumbar levels. This group predominantly shows moderate degeneration extending throughout the lumbar spine. Notably, Group H.3 also presents the greatest disc deterioration at the most cranial lumbar level (L1-L2), where approximately 70% of patients exhibit Grade III degeneration. Although herniations are located at the two lowest segments, degeneration is evident along the entire lumbar spine, with moderate degeneration (Grade III) present in all discs and more advanced degeneration (Grades IV-V) observed at the fourth and fifth lumbar levels.

Overall, the patterns demonstrate that degeneration is most severe at the herniated levels but frequently extends beyond them, with lower lumbar segments showing the greatest deterioration and Group H.3 displays the widest spread of moderate-to-advanced disc degeneration across the entire lumbar spine.

4.6.4. Pfirrmann Classification vs IVD Height

In addition to the analysis of the Pfirrmann classification, the relationship between this classification and anterior and posterior intervertebral disc heights was also examined (Tables 4.30 to 4.35).

Table 4.30 - Difference in IVD height in all Groups

	L1-L2 to L2-L3		L2-L3 to L3-L4		L3-L4 to L4-L5		L4-L5 to L5-S1	
	A	P	A	P	A	P	A	P
Group H.1	8%	15%	5%	-3%	13%	19%	37%	-10%
Group H.2	15%	13%	16%	5%	19%	3%	-7%	-23%
Group H.3	12%	12%	15%	8%	11%	-8%	-3%	-8%

A - Anterior; P - Posterior.

Table 4.31 - Frequency of distribution of Pfirrmann Classification of Group H.1.

	L1-L2	L2-L3	L3-L4	L4-L5	L5-S1
I	0	0	0	0	0
II	3	4	2	1	3
III	3	3	4	3	2
IV	1	0	1	3	2
V	0	0	0	0	0

Table 4.32 - Frequency of distribution of Pfirrmann Classification of Group H.2.

	L1-L2	L2-L3	L3-L4	L4-L5	L5-S1
I	2	8	1	1	0
II	3	6	3	0	0
III	2	6	2	2	0
IV	2	3	5	2	0
V	0	0	4	6	2

Table 4.33 - Frequency of distribution of Pfirrmann Classification of Group H.3.

	L1-L2	L2-L3	L3-L4	L4-L5	L5-S1
I	0	0	2	1	0
II	2	3	0	1	0
III	5	3	4	2	3
IV	0	1	1	3	3
V	0	0	0	0	1

In Group H.1, posterior height reduction appears at L3-L4, while the herniated level (L4-L5) does not show evidence of disc height collapse. Instead, L4-L5 presents height values that remain within the expected range for this segment, despite exhibiting higher Pfirrmann grades.

The most pronounced height alteration occurs at the inferior adjacent level (L5-S1), which shows a marked increase in anterior height and a reduction in posterior height. This indicates that, in this group, the degenerative process is more clearly reflected in the adjacent segment rather than in the herniated disc itself. Group H.2 shows the most severe degenerative pattern, with the greatest posterior height reduction at L5-S1 and the highest prevalence of Pfirrmann Grades IV-V. Upper lumbar levels remain structurally preserved, demonstrating highly localized degeneration. Group H.3 exhibits a broader pattern, with moderate upper-level association and posterior height loss at both herniated levels, accompanied by higher Pfirrmann grades. This suggests that multiple herniations promote increasing, multi-level degeneration rather than isolated structural changes.

Across all groups, a clear distinction is observed between degeneration at the herniated discs and at the adjacent levels. The herniated segments consistently show the highest Pfirrmann grades and the most evident reductions in posterior disc height, indicating that degeneration is most advanced at the symptomatic level. In contrast, adjacent discs generally preserve the height and exhibit milder degenerative changes, suggesting that degeneration remains predominantly localised rather than evenly distributed along the lumbar spine. Consistent with biomechanical principles described by Martin et al. 2002, who reported that the lower lumbar segments sustain the highest mechanical loading and therefore present more advanced degeneration, the present findings show a clear distinction between degeneration at herniated discs and at their adjacent levels [57]. It is also reflected in the modified Pfirrmann grading approach proposed by Griffith et al., 2007, in which greater posterior height reduction corresponds to higher degenerative severity. [58]. In all groups, the herniated levels consistently demonstrate the highest Pfirrmann grades and the most pronounced posterior height loss, indicating advanced degeneration directly at the symptomatic segments. In contrast, adjacent levels generally maintain disc height and display milder degenerative features, reinforcing the notion that degeneration in LDH tends to remain localised to the affected level rather than extending uniformly across adjacent discs.

In Group H.1, the herniated level (L4-L5) shows advanced degeneration without clear disc height collapse, while more evident height alterations are seen at the adjacent levels, particularly inferiorly at L5-S1. Group H.2 concentrates degeneration at the herniated disc with preservation of upper levels, and Group H.3 exhibits broader involvement but with the most pronounced changes still centred on the herniated segments. These results reinforce the close relationship between mechanical loading, disc height alterations and degenerative severity in LDH.

CHAPTER 5

CONCLUSIONS

The findings of this work are summarised in this final chapter. The investigation on LDH still has many questions to be answered, so the limitations identified across the work and future research lines are also listed.

5.1. Conclusions

The main purpose of this study was to determine how LDH at one or both of the two lowest lumbar levels affects the morphology and biomechanics of adjacent discs. By combining quantitative MRI morphometry with Pfirrmann grading, this work provided a detailed characterisation of structural behaviour at symptomatic and adjacent segments.

The analysis of IVD height showed that the herniated levels consistently presented the most marked alterations, particularly a reduction in posterior disc height, consistent with the degenerative pattern described in previous MRI studies on LDH. When compared with reference values from healthy populations, specifically the study including 171 normal individuals aged 12-80 years, the herniated discs deviated clearly from the expected anatomical pattern, whereas the discs immediately above them displayed only small and irregular variations, without a consistent trend across groups. Correlations with age supported findings from prior work showing age-related disc height loss occurring in a non-uniform pattern across the lumbar spine [51], but this effect was strongest in patients with two-level herniation rather than in the adjacent discs of single-level LDH. BMI also showed moderate associations with disc height, based on the correlations obtained in this study, although this effect did not intensify at the adjacent levels. Patient height showed no significant correlation with IVD height based on the results obtained in this study. Overall, disc height effectively identified degeneration at the herniated level but did not demonstrate structural involvement of the superior adjacent discs.

The analysis of vertebral height revealed gradual age-related reductions, consistent with the age-related tendencies reported in anatomical studies of the lumbar spine. BMI demonstrated a moderate association with vertebral dimensions, although without particular emphasis on adjacency to a herniated disc. These findings confirm that vertebral height behaves as a stable anatomical parameter and does not provide evidence of adjacent-level effects caused by LDH.

The results for the LLA showed that patients with L4-L5 herniation consistently exhibited reduced lordosis, even when an additional herniation at L5-S1 was present. This pattern aligns with previous observations that disc degeneration and reduced disc height at L4-L5 are associated with lower lumbar curvature. In contrast, patients with isolated L5-S1 herniation demonstrated lordosis values closer to those reported in asymptomatic adults. Although age and BMI influenced lordosis, these variations were not accompanied by measurable changes in the

adjacent discs, showing that LLA varies independently of degeneration at the levels above the herniation.

The intervertebral angles in the sagittal and coronal planes demonstrated reductions predominantly at the herniated levels. The behaviour of the adjacent levels was more variable, and the high measurement variability, especially in the coronal plane, limited the interpretability of the results. Although angular reduction in LDH is consistent with decreased disc height described in biomechanical studies, no systematic alteration appeared in the discs above the herniation. Thus, angular measures highlighted local consequences of LDH but did not reveal adjacent-level association.

The Pfirrmann classification provided the clearest evidence regarding adjacent-level degeneration. The herniated discs showed the highest grades of degeneration, confirming the localised structural damage described in the literature. Importantly, the discs immediately superior to the herniated segment often exhibited higher degeneration grades than the more cranial levels, even when height measurements remained preserved. This pattern was particularly evident in patients with L4-L5 herniation, where the L3-L4 disc frequently demonstrated moderate degeneration. In patients with two-level herniation, the degenerative pattern was broader meaning that multi-level pathology is associated with more extensive degenerative distribution. These observations confirm that when applying Pfirrmann classification, degenerative influence extending into the adjacent superior disc is noticeable, differing from the outputs of quantitative morphometry. This is consistent with imaging studies demonstrating that qualitative MRI grading can reveal early degenerative changes not yet reflected in disc height or angular measurements.

In summary, LDH produced marked structural alterations at the symptomatic levels, reflected in disc height reduction, altered angles and advanced Pfirrmann grades. Quantitative MRI parameters, although altered, did not demonstrate a consistent alteration trend in the superior adjacent discs. However, the Pfirrmann classification showed a moderate but clear increase in degenerative grade at the discs immediately above the herniated segment, providing consistent evidence of adjacent-level association. Thus, while LDH does not induce measurable structural collapse in the segments above, it contributes to detectable degenerative changes identifiable through qualitative MRI assessment.

5.2. Limitations

The study is subject to several methodological and technical limitations. First, all MRI exams were acquired in a standard 2D clinical setting, which restricts volumetric accuracy and excludes direct three-dimensional measurement of anatomical structures. In addition, most exams did not include a coronal plane, requiring MPR to perform certain measurements. Although MPR enabled the visualization of the missing plane, it inherently reduced image resolution, likely contributing to the elevated deviations observed between some of the measures.

Manual quantification also posed challenges, particularly for angular and area-based parameters, which depend heavily on the observer’s interpretation of anatomical boundaries.

Despite the use of repeated measurements to minimize bias, inter- and intra-observer variability remained considerable. These procedures are not only time-consuming but also difficult to reproduce, reflecting a broader limitation of manual MRI analysis in clinical research.

A further limitation arises from the use of the Pfirrmann classification, which is essentially qualitative and relies on visual interpretation of disc signal and morphology. As such, it is vulnerable to subjective judgement and may introduce classification errors, especially in borderline or intermediate grades.

Moreover, the relatively small sample limited the statistical power of correlations. Finally, the cross-sectional design of the study restricted the ability to evaluate temporal progression or causal relationships between structural degeneration and functional impairment, although if a longitudinal study were possible, patients would have to undergo MRI scans again.

5.3. Future Work

To overcome these limitations, future work should focus on expanding the study sample and incorporating balanced demographic variables, including sex-based analyses that may reveal subtle anatomical and biomechanical differences. The acquisition of true 3D MRI datasets would possibly enable more precise and reproducible morphometric analysis, reducing dependence on reconstructed planes.

An additional priority should be the development of automated systems for measurement extraction and parameter quantification. Integrating artificial intelligence and deep learning approaches could automate segmentation, classification, and measurement processes, mitigating human error and significantly reducing analysis time. In this context, developing an MRI-based method, as previously presented, capable of reliably identifying and characterising lumbar IVD’s would be particularly valuable, as it would allow accurate, large-scale characterisation of disc morphology and degeneration that is not feasible through manual analysis alone. These tools could also facilitate large-scale cohort studies by processing high volumes of MRI data rapidly and consistently.

Correlating imaging-derived morphometric data with functional outcomes, such as pain intensity, and clinical recovery trajectories, would provide a better understanding of the biomechanical and clinical impact of LDH. Finally, incorporating patient-specific computational models through FEA would offer predictive understandings into spinal loading dynamics, degeneration risk, and personalized surgical planning.

Such advancements will consolidate the connection between imaging, biomechanics, and clinical application, enabling a fully integrated, quantitative, and patient-specific structure for understanding and managing LDH.

REFERENCES

[1] J. S. Will, D. C. Bury, and J. A. Miller, “Mechanical Low Back Pain.,” *Am Fam Physician*, vol. 98, no. 7, pp. 421–428, Oct. 2018.

[2] C. E. Alexander, L. J. Weisbrod, and M. A. Varacallo, *Lumbosacral Radiculopathy*. 2025.

[3] M. I. Al Qaraghli and O. De Jesus, “Lumbar Disc Herniation,” https://www.ncbi.nlm.nih.gov/books/NBK560878/?report=classic.

[4] R. Läubli *et al.*, “Disproportionate Vertebral Bodies and Their Impact on Lumbar Disc Herniation,” *J Clin Med*, vol. 10, no. 14, p. 3174, Jul. 2021, doi: 10.3390/jcm10143174.

[5] A. S. Zhang *et al.*, “Lumbar Disc Herniation: Diagnosis and Management,” *Am J Med*, vol. 136, no. 7, pp. 645–651, Jul. 2023, doi: 10.1016/j.amjmed.2023.03.024.

[6] P. Roussouly and J. L. Pinheiro-Franco, “Sagittal parameters of the spine: biomechanical approach,” *European Spine Journal*, vol. 20, no. S5, pp. 578–585, Sep. 2011, doi: 10.1007/s00586-011-1924-1.

[7] P. J. Bazira, “Clinically applied anatomy of the vertebral column,” *Surgery (Oxford)*, vol. 39, no. 6, pp. 315–323, Jun. 2021, doi: 10.1016/j.mpsur.2021.04.004.

[8] V. Mahadevan, “Anatomy of the vertebral column,” *Surgery (Oxford)*, vol. 36, no. 7, pp. 327–332, Jul. 2018, doi: 10.1016/j.mpsur.2018.05.006.

[9] N. Bogduk, “Functional anatomy of the spine,” 2016, pp. 675–688. doi: 10.1016/B978-0-444-53486-6.00032-6.

[10] B. Sassack and J. Carrier, “Anatomy, Back, Lumbar Spine,” https://www.ncbi.nlm.nih.gov/books/NBK557616/.

[11] N. A. Ebraheim, A. Hassan, M. Lee, and R. Xu, “Functional anatomy of the lumbar spine,” *Seminars in Pain Medicine*, vol. 2, no. 3, pp. 131–137, Sep. 2004, doi: 10.1016/j.spmd.2004.08.004.

[12] M. Gallucci, E. Puglielli, A. Splendiani, F. Pistoia, and G. Spacca, “Degenerative disorders of the spine,” *Eur Radiol*, vol. 15, no. 3, pp. 591–598, Mar. 2005, doi: 10.1007/s00330-004-2618-4.

[13] J.-P. Valat, S. Genevay, M. Marty, S. Rozenberg, and B. Koes, “Sciatica,” *Best Pract Res Clin Rheumatol*, vol. 24, no. 2, pp. 241–252, Apr. 2010, doi: 10.1016/j.berh.2009.11.005.

[14] J.-P. Valat, S. Genevay, M. Marty, S. Rozenberg, and B. Koes, “Sciatica,” *Best Pract Res Clin Rheumatol*, vol. 24, no. 2, pp. 241–252, Apr. 2010, doi: 10.1016/j.berh.2009.11.005.

[15] C. Jin, S. Wang, G. Yang, E. Li, and Z. Liang, “A Review of the Methods on Cobb Angle Measurements for Spinal Curvature,” *Sensors*, vol. 22, no. 9, p. 3258, Apr. 2022, doi: 10.3390/s22093258.

[16] G. S. Skaf, C. M. Ayoub, N. T. Domloj, M. J. Turbay, C. El-Zein, and M. H. Hourani, “Effect of Age and Lordotic Angle on the Level of Lumbar Disc Herniation,” *Adv Orthop*, vol. 2011, pp. 1–6, 2011, doi: 10.4061/2011/950576.

[17] S. Genevay and S. J. Atlas, “Lumbar Spinal Stenosis,” *Best Pract Res Clin Rheumatol*, vol. 24, no. 2, pp. 253–265, Apr. 2010, doi: 10.1016/j.berh.2009.11.001.

[18] C. S. Lee *et al.*, “Risk factors for adjacent segment disease after lumbar fusion,” *European Spine Journal*, vol. 18, no. 11, pp. 1637–1643, Nov. 2009, doi: 10.1007/s00586-009-1060-3.

[19] P. K. Louie *et al.*, “Etiology-Based Classification of Adjacent Segment Disease following Lumbar Spine Fusion,” *HSS J*, vol. 16, no. 2, pp. 130–136, Jul. 2020, doi: 10.1007/s11420-019-09723-w.

[20] O. Tsuji *et al.*, “Effectiveness of Duloxetine for Postsurgical Chronic Neuropathic Disorders after Spine and Spinal Cord Surgery,” *Asian Spine J*, vol. 15, no. 5, pp. 650–658, Oct. 2021, doi: 10.31616/asj.2020.0191.

[21] S. V. Kushchayev *et al.*, “ABCs of the degenerative spine,” *Insights Imaging*, vol. 9, no. 2, pp. 253–274, Apr. 2018, doi: 10.1007/s13244-017-0584-z.

[22] D. F. Fardon, A. L. Williams, E. J. Dohring, F. R. Murtagh, S. L. Gabriel Rothman, and G. K. Sze, “Lumbar disc nomenclature: version 2.0,” *The Spine Journal*, vol. 14, no. 11, pp. 2525–2545, Nov. 2014, doi: 10.1016/j.spinee.2014.04.022.

[23] C. H. Oh and S. H. Yoon, “Whole Spine Disc Degeneration Survey according to the Ages and Sex Using Pfirrmann Disc Degeneration Grades,” *Korean J Spine*, vol. 14, no. 4, pp. 148–154, Dec. 2017, doi: 10.14245/kjs.2017.14.4.148.

[24] D. S. Kreiner *et al.*, “An evidence-based clinical guideline for the diagnosis and treatment of lumbar disc herniation with radiculopathy,” *The Spine Journal*, vol. 14, no. 1, pp. 180–191, Jan. 2014, doi: 10.1016/j.spinee.2013.08.003.

[25] M. C. Bicket, K. Chakravarthy, D. Chang, and S. P. Cohen, “Epidural Steroid Injections: An Updated Review on Recent Trends in Safety and Complications,” *Pain Manag*, vol. 5, no. 2, pp. 129–146, Mar. 2015, doi: 10.2217/pmt.14.53.

[26] R. A. Deyo, S. J. Bigos, and K. R. Maravilla, “Diagnostic Imaging Procedures for the Lumbar Spine,” *Ann Intern Med*, vol. 111, no. 11, pp. 865–867, Dec. 1989, doi: 10.7326/0003-4819-111-11-865.

[27] J. N. A. Gibson and G. Waddell, “Surgical Interventions for Lumbar Disc Prolapse,” *Spine (Phila Pa 1976)*, vol. 32, no. 16, pp. 1735–1747, Jul. 2007, doi: 10.1097/BRS.0b013e3180bc2431.

[28] S. J. Kamper *et al.*, “Minimally invasive surgery for lumbar disc herniation: a systematic review and meta-analysis,” *European Spine Journal*, Jan. 2014, doi: 10.1007/s00586-013-3161-2.

[29] S. Ruetten, M. Komp, H. Merk, and G. Godolias, “Full-Endoscopic Interlaminar and Transforaminal Lumbar Discectomy Versus Conventional Microsurgical Technique,” *Spine (Phila Pa 1976)*, vol. 33, no. 9, pp. 931–939, Apr. 2008, doi: 10.1097/BRS.0b013e31816c8af7.

[30] M. Teli *et al.*, “Higher risk of dural tears and recurrent herniation with lumbar micro-endoscopic discectomy,” *European Spine Journal*, vol. 19, no. 3, pp. 443–450, Mar. 2010, doi: 10.1007/s00586-010-1290-4.

[31] J. N. Weinstein *et al.*, “Surgical Versus Nonoperative Treatment for Lumbar Disc Herniation,” *Spine (Phila Pa 1976)*, vol. 33, no. 25, pp. 2789–2800, Dec. 2008, doi: 10.1097/BRS.0b013e31818ed8f4.

[32] J.-H. Kim *et al.*, “Diagnostic accuracy of diagnostic imaging for lumbar disc herniation in adults with low back pain or sciatica is unknown; a systematic review,” *Chiropr Man Therap*, vol. 26, no. 1, p. 37, Dec. 2018, doi: 10.1186/s12998-018-0207-x.

[33] P. C. Milette, “Classification, Diagnostic Imaging, and Characterization of a Lumbar Herniated Disc,” *Radiol Clin North Am*, vol. 38, no. 6, pp. 1267–1292, Nov. 2000, doi: 10.1016/S0033-8389(08)70006-X.

[34] A. M. Dydyk, R. N. Massa, and F. B. Mesfin, “Disc Herniation,” https://www.ncbi.nlm.nih.gov/books/NBK441822/.

[35] T. Çetin, S. Kahraman, V. Kızılgöz, and S. Aydın, “The Comparison between Herniated and Non-Herniated Disc Levels Regarding Intervertebral Disc Space Height and Disc Degeneration, A Magnetic Resonance Study,” *Diagnostics*, vol. 13, no. 20, p. 3190, Oct. 2023, doi: 10.3390/diagnostics13203190.

[36] A. Tunset, P. Kjaer, S. Samir Chreiteh, and T. Secher Jensen, “A method for quantitative measurement of lumbar intervertebral disc structures: an intra- and inter-rater agreement and reliability study,” *Chiropr Man Therap*, vol. 21, no. 1, p. 26, Dec. 2013, doi: 10.1186/2045-709X-21-26.

[37] M. Pojskic, E. Bisson, J. Oertel, T. Takami, C. Zygourakis, and F. Costa, “Lumbar disc herniation: Epidemiology, clinical and radiologic diagnosis WFNS spine committee recommendations,” *World Neurosurg X*, vol. 22, p. 100279, Apr. 2024, doi: 10.1016/j.wnsx.2024.100279.

[38] A. Depeursinge, A. Foncubierta-Rodriguez, D. Van De Ville, and H. Müller, “Three-dimensional solid texture analysis in biomedical imaging: Review and opportunities,” *Med Image Anal*, vol. 18, no. 1, pp. 176–196, Jan. 2014, doi: 10.1016/j.media.2013.10.005.

[39] N. Salsabili, J. Santiago López, and M. I. Prieto Barrio, “Simplifying the human lumbar spine (L3/L4) material in order to create an elemental structure for the future modeling,” *Australas Phys Eng Sci Med*, vol. 42, no. 3, pp. 689–700, Sep. 2019, doi: 10.1007/s13246-019-00768-z.

[40] S. Wang, Z. Jiang, H. Yang, X. Li, and Z. Yang, “Automatic Segmentation of Lumbar Spine MRI Images Based on Improved Attention U-Net,” *Comput Intell Neurosci*, vol. 2022, pp. 1–10, Sep. 2022, doi: 10.1155/2022/4259471.

[41] M. Ahmadi *et al.*, “Integrating finite element analysis and physics-informed neural networks for biomechanical modeling of the human lumbar spine,” *North American Spine Society Journal (NASSJ)*, vol. 22, p. 100598, Jun. 2025, doi: 10.1016/j.xnsj.2025.100598.

[42] J. Schlemper *et al.*, “Attention gated networks: Learning to leverage salient regions in medical images,” *Med Image Anal*, vol. 53, pp. 197–207, Apr. 2019, doi: 10.1016/j.media.2019.01.012.

[43] J. Chen, L. Qian, L. Ma, T. Urakov, W. Gu, and L. Liang, “SymTC: A symbiotic Transformer-CNN net for instance segmentation of lumbar spine MRI,” *Comput Biol Med*, vol. 179, p. 108795, Sep. 2024, doi: 10.1016/j.compbiomed.2024.108795.

[44] E. E.-Y. Yilihamu *et al.*, “Quantification and classification of lumbar disc herniation on axial magnetic resonance images using deep learning models,” *Radiol Med*, vol. 130, no. 6, pp. 795–804, Mar. 2025, doi: 10.1007/s11547-025-01996-y.

[45] B. Rostami, D. M. Anisuzzaman, C. Wang, S. Gopalakrishnan, J. Niezgoda, and Z. Yu, “Multiclass wound image classification using an ensemble deep CNN-based classifier,” *Comput Biol Med*, vol. 134, p. 104536, Jul. 2021, doi: 10.1016/j.compbiomed.2021.104536.

[46] A. P. G. Castro, P. Flores, J. C. P. Claro, A. M. G. Completo, and J. L. Alves, “On the Computational Biomechanics of the Intervertebral Disc,” 2020, pp. 223–240. doi: 10.1007/978-3-030-37541-6_9.

[47] R. Matos, “Segmentação de discos intervertebrais lombares para modelação e simulação computacional,” FACULDADE DE CIÊNCIAS - UNIVERSIDADE DE LISBOA , Lisbon, 2021.

[48] “Obesity: preventing and managing the global epidemic. Report of a WHO consultation.,” *World Health Organ Tech Rep Ser*, vol. 894, pp. i–xii, 1–253, 2000.

[49] I. F. Mendes, “Characterisation and Assessment of Lumbar Disc Herniation using Image Segmentation and Modelling,” Instituto Politécnico de Setúbal - Escola Superior de Tecnologia de Setúbal, Setúbal , 2024.

[50] Spencer B. GAY, Norman C. Chen, Joseph J. Burch, Thomas R. Gleason, and Arthur M. Sagman, “Multiplanar Reconstruction in Magnetic Resonance Evaluation of the Knee”.

[51] I. Malkoç *et al.*, “Age Related Changes in Height and Shape of the Lumbar İntervertebral Discus,” *European Journal of Basic Medical Sciences*, vol. 2, pp. 68–73, Sep. 2012, doi: 10.21601/ejbms/9182.

[52] M. A. Adams and P. J. Roughley, “What is Intervertebral Disc Degeneration, and What Causes It?,” *Spine (Phila Pa 1976)*, vol. 31, no. 18, pp. 2151–2161, Aug. 2006, doi: 10.1097/01.brs.0000231761.73859.2c.

[53] N. K. Singh, N. K. Singh, R. Verma, and A. D. Diwan, “Validation and Estimation of Obesity-Induced Intervertebral Disc Degeneration through Subject-Specific Finite Element Modelling of Functional Spinal Units,” *Bioengineering*, vol. 11, no. 4, p. 344, Mar. 2024, doi: 10.3390/bioengineering11040344.

[54] S. M. H. Mirab, M. Barbarestani, S. M. Tabatabaei, S. Shahsavari, and M. B. Minaeii Zangi, “Measuring Dimensions of Lumbar Intervertebral Discs in Normal Subjects,” *ASJ*, vol. 15, no. 1, pp. 3–8, Feb. 2018, [Online]. Available: http://anatomyjournal.ir/article-1-197-en.html

[55] J.-R. Mi Le, K.-T. Yeh, C.-W. Chen, F.-S. Jaw, S.-H. Yang, and W.-T. Wu, “Quantitative evaluation of correlation between lumbosacral lordosis and pelvic incidence in standing position among asymptomatic Asian adults: a prospective study,” *Sci Rep*, vol. 12, no. 1, p. 18965, Nov. 2022, doi: 10.1038/s41598-022-21840-x.

[56] H. Zhou *et al.*, “L4/5 Disc Herniation: Not Unusually Accompanied with L5/S1 Low-Grade Spondylolytic Spondylolisthesis,” *Orthop Surg*, vol. 16, no. 2, pp. 444–451, Feb. 2024, doi: 10.1111/os.13984.

[57] M. D. Martin, C. M. Boxell, and D. G. Malone, “Pathophysiology of lumbar disc degeneration: a review of the literature,” *Neurosurg Focus*, vol. 13, no. 2, pp. 1–6, Aug. 2002, doi: 10.3171/foc.2002.13.2.2.

[58] J. F. Griffith *et al.*, “Modified Pfirrmann Grading System for Lumbar Intervertebral Disc Degeneration,” *Spine (Phila Pa 1976)*, vol. 32, no. 24, pp. E708–E712, Nov. 2007, doi: 10.1097/BRS.0b013e31815a59a0.

APPENDIX A

CLINICAL PARAMETERS OF THE PATIENTS IN THE DATABASE

Table A.1- Clinical and Demographic from the patients.

Code	Age (years)	Sex	Weight (kg)	Height (cm)	BMI (kg/m²)	BMI Classification	Level of Herniation
1	55	M	85	171	29,1	Overweight	L4-L5 and L5-S1
2	48	F	63	156	25,9	Overweight	L5-S1
3	51	M	127	NA	38,3	Obesity Grade II	L4-L5 and L5-S1
4	66	F	85	167	30,5	Obesity Grade I	L4-L5 and L5-S1
5	28	F	55	165	20,2	Normal Weight	L5-S1
6	43	F	76	167	27,3	Overweight	L5-S1
7	28	M	74	174	24,4	Normal Weight	L4-L5 and L5-S1
8	43	M	97	177	31,0	Obesity Grade I	L4-L5
9	37	M	80	188	22,6	Normal Weight	L4-L5
10	37	M	74	175	24,2	Normal Weight	L5-S1
11	50	F	57	150	25,3	Overweight	L5-S1
12	57	M	65	159	25,7	Overweight	L4-L5 and L5-S1
13	77	M	72	72	24,9	Normal Weight	L5-S1
14	71	M	83	165	30,5	Obesity Grade I	L4-L5
15	61	M	93	163	35,0	Obesity Grade II	L4-L5 and L5-S1
16	27	M	95	183	28,4	Overweight	L4-L5 and L5-S1
17	58	M	71	170	24,6	Normal Weight	L4-L5
18	57	M	80	167	28,7	Overweight	L4-L5
19	60	F	90	157	36,5	Obesity Grade II	L5-S1
20	30	M	90	178	28,4	Overweight	L5-S1
21	64	F	75	160	29,3	Overweight	L5-S1
22	25	F	46	156	18,9	Normal Weight	L5-S1
23	49	F	102	167	36,6	Obesity Grade II	L5-S1
24	44	F	99	156	40,7	Obesity Grade III	L4-L5
25	60	F	72	166	26,1	Overweight	L5-S1
26	58	M	72	170	24,9	Normal Weight	L4-L5

NA - not available

APPENDIX B

AVERAGE MEASUREMENTS OF THE PRESENT STUDY

A. IVD HEIGHT

Table B.1 - Average Anterior, Central and Posterior measurements of IVD Height.

Average IVD height										
Code	L1-L2 (mm)		L2-L3 (mm)		L3-L4 (mm)		L4-L5 (mm)		L5-S1 (mm)	
	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior
1	10,32	5,58	10,34	5,23	12,25	7,52	11,71	6,64	10,57	5,29
2	6,94	6,47	9,09	6,97	7,54	8,40	11,30	9,42	5,96	5,42
3	9,45	4,85	12,38	5,92	11,97	7,02	12,47	4,30	8,57	5,38
4	9,00	7,85	10,20	8,45	12,29	7,17	13,21	5,49	14,25	6,44
5	6,52	5,59	8,15	7,06	9,73	7,95	13,68	7,43	15,28	6,46
6	8,52	8,05	9,91	8,94	12,38	8,88	12,72	7,90	11,16	6,71
7	8,72	4,78	8,94	5,50	11,53	6,25	13,38	7,02	13,43	6,60
8	9,62	6,87	11,13	7,03	13,53	8,11	14,13	10,40	17,02	9,78
9	10,69	5,12	10,97	6,05	11,43	7,15	13,54	9,46	17,52	8,20
10	8,85	5,15	9,32	4,84	10,43	6,14	12,14	6,81	11,46	5,00
11	8,73	7,94	9,48	7,24	9,77	6,33	11,26	7,60	11,45	6,82
12	7,59	9,09	10,06	9,94	9,91	9,49	14,76	9,58	15,26	7,51
13	7,52	5,07	5,68	5,68	9,21	7,06	11,18	7,30	13,93	6,12
14	10,34	6,50	11,04	8,67	8,32	6,69	13,75	8,73	15,36	8,08

Average IVD height										
Code	L1-L2 (mm)		L2-L3 (mm)		L3-L4 (mm)		L4-L5 (mm)		L5-S1 (mm)	
	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior
15	8,94	6,79	9,08	7,86	10,74	7,97	8,91	7,28	12,57	7,90
16	9,72	8,01	10,23	9,73	12,75	9,77	15,46	11,31	9,98	4,66
17	10,17	7,15	9,74	8,11	10,28	7,39	11,95	7,86	11,18	5,27
18	9,74	8,15	11,38	9,58	12,91	8,99	10,19	8,88	18,54	10,72
19	7,99	6,60	9,27	7,10	9,86	6,82	11,98	7,01	11,35	4,91
20	6,47	5,37	8,77	8,81	11,06	8,65	13,81	9,13	14,06	7,19
21	7,79	7,36	8,99	6,99	9,97	7,10	10,00	6,12	6,70	3,92
22	7,71	5,02	10,42	6,93	10,63	7,83	13,51	7,73	16,18	7,39
23	9,43	9,62	9,60	9,58	12,07	8,97	13,00	9,45	5,58	4,37
24	6,98	6,56	8,47	7,06	9,95	7,81	12,46	8,50	16,75	7,86
25	7,84	7,59	9,42	8,19	11,01	7,14	11,57	7,56	13,89	6,59
26	10,19	10,16	9,74	11,31	9,82	8,16	7,74	10,48	14,69	8,30

B. VERTEBRAE HEIGHT

Table B.2 - Average Anterior, Central and Posterior measurements of Vertebrae Height.

Average vertebrae height												
Code	L1 (mm)		L2 (mm)		L3 (mm)		L4 (mm)		L5 (mm)		S1 (mm)	
	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior
1	25,22	28,95	27,10	30,95	27,07	30,21	29,02	29,39	31,03	23,40	35,77	27,43
2	24,28	22,80	26,36	23,26	28,94	24,44	29,41	23,54	27,28	20,16	30,06	20,14
3	25,56	27,98	27,05	30,37	30,14	28,01	25,89	28,93	30,60	23,36	27,92	26,67
4	24,33	25,78	25,89	26,43	26,65	27,47	26,21	25,54	27,95	23,96	32,72	25,46
5	23,54	24,66	25,11	26,36	27,40	26,15	26,87	25,18	26,85	23,19	28,91	20,84
6	25,25	26,53	26,62	26,26	28,60	25,72	25,00	24,01	24,94	24,17	32,51	23,72
7	26,97	30,86	27,36	30,32	29,27	32,15	28,60	30,08	27,32	26,57	30,78	24,38
8	26,25	29,17	24,88	28,36	26,67	30,39	25,36	26,88	27,81	23,64	33,53	25,95
9	22,77	28,39	27,64	29,39	27,88	30,47	26,81	26,13	25,88	25,34	32,62	23,76
10	27,00	27,79	27,13	28,85	27,44	29,04	27,33	28,44	27,58	26,21	30,86	26,29
11	24,00	23,25	26,42	24,14	24,58	24,26	23,79	22,01	24,16	20,35	28,34	22,32
12	24,86	23,77	24,22	24,53	25,70	23,89	25,20	22,97	26,15	19,41	31,18	25,28
13	26,30	26,40	25,11	28,45	27,69	26,68	28,21	25,46	29,57	24,07	33,14	24,24
14	22,69	28,64	23,12	27,71	27,72	26,84	25,50	23,56	26,61	20,05	30,09	20,30
15	24,73	25,73	25,04	26,17	25,77	25,49	25,69	24,48	24,30	21,79	30,20	25,65
16	25,72	29,13	27,97	27,67	29,07	28,11	27,43	27,22	29,94	25,51	34,87	24,46
17	26,51	24,44	27,76	26,34	25,87	24,88	26,99	23,28	27,00	20,91	34,96	29,72
18	23,94	26,02	25,43	26,95	26,52	26,33	24,09	25,45	23,51	23,08	32,27	23,59
19	23,37	21,84	25,76	23,69	23,89	24,68	23,98	24,90	23,99	22,78	27,21	21,14

Average vertebrae height												
Code	L1 (mm)		L2 (mm)		L3 (mm)		L4 (mm)		L5 (mm)		S1 (mm)	
	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior	Anterior	Posterior
20	26,86	27,50	27,79	28,73	27,55	28,40	30,31	27,19	31,34	21,71	34,81	29,84
21	22,34	23,59	26,57	25,59	25,53	24,23	25,08	22,73	25,17	22,31	32,73	24,69
22	23,19	25,04	22,86	26,73	25,76	26,51	26,23	25,95	26,48	21,61	26,27	21,28
23	23,09	23,66	25,16	23,13	26,02	24,29	24,07	23,47	21,85	21,59	31,96	22,94
24	22,99	25,21	24,41	25,09	27,70	23,89	24,20	22,97	25,59	21,56	30,67	23,98
25	24,66	26,68	25,91	26,47	27,43	24,39	27,08	23,57	28,20	22,64	32,14	25,36
26	25,49	25,40	26,37	24,13	27,65	25,09	27,22	23,99	28,52	20,87	33,28	25,21

C. INTERVERTEBRAL ANGLE

Table B.3 - Average measurements of Intervertebral Angle in Sagittal and Coronal Planes.

Average Intervertebral Angle (º)										
Code	**Sagittal Plane**					**Coronal Plane**				
	L1-L2	**L2-L3**	**L3-L4**	**L4-L5**	**L5-S1**	**L1-L2**	**L2-L3**	**L3-L4**	**L4-L5**	**L5-S1**
1	7,85	7,41	9,41	9,30	8,63	1,12	0,25	0,81	1,25	2,40
2	0,45	3,43	4,79	6,54	4,34	1,63	1,28	1,76	0,71	4,45
3	4,09	5,32	5,59	12,09	8,46	2,67	2,45	3,04	3,32	3,07
4	2,29	3,11	7,21	14,05	18,06	3,05	3,76	1,35	4,65	1,15
5	3,38	4,63	4,89	11,12	20,41	3,92	3,16	1,16	0,94	0,85
6	1,28	1,21	6,70	8,25	13,36	1,89	1,30	2,77	0,54	1,69
7	8,54	6,83	10,92	10,18	15,33	1,27	0,92	0,37	1,34	0,76
8	3,32	5,26	5,50	1,79	13,48	1,11	1,60	0,48	0,31	1,44
9	6,80	8,37	8,35	5,35	8,79	0,70	0,19	1,12	1,61	1,62
10	4,61	4,43	7,26	11,26	14,71	0,85	0,64	1,00	1,35	0,44
11	1,48	5,71	6,43	7,89	11,51	2,44	0,55	1,35	2,41	1,27
12	2,85	0,52	1,55	12,43	15,65	1,05	1,48	0,65	0,79	1,41
13	4,01	1,41	3,60	8,97	16,16	1,27	1,29	0,29	0,85	1,76
14	5,50	4,44	4,90	11,35	15,28	0,85	2,02	0,89	4,16	1,94
15	3,82	2,81	4,23	5,73	12,83	0,50	1,52	0,35	0,17	0,48
16	4,00	0,97	5,17	9,04	11,85	1,27	0,94	2,26	1,80	0,77
17	7,03	5,02	7,75	7,68	12,49	1,70	1,18	1,52	2,05	2,33
18	3,75	5,35	7,06	2,92	14,91	1,04	1,22	0,56	0,62	1,56
19	7,12	6,61	8,61	9,45	14,77	1,07	0,32	0,75	1,97	1,76
20	2,59	1,34	3,91	10,84	17,50	1,81	2,05	1,01	0,49	1,15
21	2,24	4,38	7,24	9,33	8,94	0,30	0,23	1,01	0,58	1,17
22	6,45	6,43	9,84	14,64	22,22	1,93	1,17	1,68	2,35	2,02
23	0,87	2,15	6,64	8,86	5,82	0,63	0,58	0,53	2,32	2,82
24	0,93	0,97	5,67	7,90	20,62	1,72	1,12	1,90	2,13	2,53
25	0,24	3,22	8,68	9,42	18,02	1,15	1,28	1,77	1,31	2,29
26	0,44	3,24	3,34	5,28	15,27	1,11	2,56	1,68	1,02	1,11

D. AVERAGE LUMBAR LORDOSIS ANGLE

Table B.4 - Average measurements of Lumbar Lordosis Angle.

Average Lumbar Lordosis Angle (LLA)	
Code	**(º)**
1	39,23
2	64,98
3	41,49
4	52,99
5	48,83
6	41,96
7	31,31
8	29,48
9	28,95
10	41,54
11	61,23
12	60,29
13	47,14
14	41,98
15	35,42
16	34,19
17	66,22
18	34,63
19	44,49
20	49,22
21	50,23
22	48,11
23	40,81
24	54,00
25	58,32
26	40,99

E. Measurements of lateral deviation

Table B.5 - Average measurements of Lateral Deviation.

Lateral Deviation	
Code	**Average**
3	2,27
4	4,70
5	1,82
8	3,78
9	1,86
10	2,75
11	2,14
12	1,50
16	3,27
17	2,01

APPENDIX C

VALUES OF REFERENCE FOR IVD HEIGHT

These Values are from the article “Age Related Changes in Height and Shape of The Lumbar Intervertebral Discus”[51].

Table C.1 - Reference values of anterior IVD height.

Table 1. Changes of anterior height of the lumbar intervertebral discs of both sexes (mm) by age groups (decades). F=female, M=male. The varied lower case in the decades indicates the differences between the age groups (a, b, c →).

	Sex	DECADES						
		10-19	20-29	30-39	40-49	50-59	60-69	70-79
		Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD
L1	F	7.94±1.75^{b}	8.56±1.08$^{\#ab}$	9.04±1,2$^{\#ab}$	9.76±1.14^{a}	9.64±.93^{a}	9.59±1,33^{a}	9.6±1.58^{a}
	M	9.68±1.17^{b}	9.83±1.61^{b}	9.89±.96^{b}	9.92±1.15^{b}	10.53±1.5^{ab}	10.16±1.1^{b}	11.46±1.9^{a}
L2	F	8.81±2.26^{b}	10.28±1.3^{ab}	10.4±1,5$^{\#ab}$	11.21±1.6^{a}	11.21±1,19^{a}	10.8±2.2^{a}	10.61±2.5^{a}
	M	10.97±1.5^{a}	11.47±2.6^{a}	11.84±1.3^{a}	11.19±1.5^{a}	10.95±1.15^{a}	11.59±1.6^{a}	12.28±1.2^{a}
L3	F	9.95±2.6^{b}	11.59±1.3^{ab}	12.35±1.19^{a}	12.49±1.51^{a}	12.45±2.22^{a}	11.57±1.8^{ab}	12.24±2.05^{a}
	M	11.66±2.14^{b}	12.41±2.42^{ab}	12.86 ±1.85^{ab}	12.79±1.93^{ab}	12.34±2.08^{ab}	12.55±1.88^{ab}	14.06±2.35^{a}
L4	F	13.33±2.97^{a}	13.6±1.36^{a}	13.49±1.99^{a}	14.09±2.01^{a}	14.80±2.27^{a}	13.72±2.41^{a}	14.53±3.35^{a}
	M	12.98±1.97^{a}	14.33±2.06^{a}	13.84±1.66^{a}	14.44±1.67^{a}	14.29 ±2.59^{a}	13.21 ±3.23^{a}	14.47±1.79^{a}
L5	F	13.55±3.24^{a}	14.45±1.6^{a}	13.74±3.96^{a}	14.38 ±3.18^{a}	16.6±3.39^{a}	15.01 ±4.32^{a}	14.4±4.82^{a}
	M	13.06±1.62^{b}	13.92±3.53^{ab}	14.17±3.32^{ab}	16.58±2.89^{a}	14.75±2.93^{ab}	16.13±3.08^{a}	16.39±4.46^{a}

: male> female is significant (p<0.05)

Table C.2 - Reference values of posterior IVD height.

Table 2. Changes of posterior height of the lumbar intervertebral discs of both sexes (mm) by age groups (decades). F=female, M=male. The varied lower case in the decades indicates the differences between the age groups (a, b, c →).

	Sex	DECADES						
		10-19	20-29	30-39	40-49	50-59	60-69	70-79
		Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD
L1	F	6.48 ±2.52^{a}	7.1 ±1.32^{a}	6.99 ±1.06^{a}	6.91±1.06^{a}	7.19±1.08^{a}	7.1±1.68^{a}	6.94±2.01^{a}
	M	7.39±1.1^{a}	6.94±1.41^{a}	6.89±1.65^{a}	6.82±1.24^{a}	6.58±1.11^{a}	6.48±0.67^{a}	6.8±1.4^{a}
L2	F	6.98±2.22^{a}	7.67±1.12^{a}	7.37±0.63^{a}	7.78±1.04^{a}	7.91±1.07^{a}	7.45±1.56^{a}	7.41±1.11^{a}
	M	8.28±0.8^{ab}	8.50±3.21^{a}	7.81±1.69^{ab}	7.2±1.03^{ab}	7.16±1.1^{ab}	7.1±0.94^{ab}	6.7±1.2^{b}
L3	F	7.43±2.05^{a}	7.91±1.23^{a}	8.32±1.15^{a}	8.01±1.35^{a}	7.82±1.25^{a}	7.57±0.67^{a}	7.97±1.42^{a}
	M	7.86±1.37^{ab}	7.97±1.29^{ab}	8.54±1.19^{a}	7.77±1.29^{ab}	7.29±1.33^{b}	7.51±1.08^{ab}	8.06±1.36^{ab}
L4	F	7.3±0.67^{a}	8.29±1.19^{a}	7.68±1.11^{a}	8.00±1.47^{a}	8.18±0.96$^{\#a}$	11.11±11.19^{a}	7.92±2.88^{a}
	M	7.35±1.12^{a}	8.08±0.95^{a}	7.94±1.66^{a}	7.57±1.31^{a}	7.35±0.76^{a}	7.1±1.3^{a}	7.98±1.61^{a}
L5	F	6.83±1.41^{a}	7.04±1.36^{a}	6.69±1.88^{a}	6.48±1.04^{a}	7.46±1.31^{a}	6.7±1.03^{a}	6.59±1.63^{a}
	M	6.73±1.50^{ab}	7.08±1.18^{ab}	6.51±1.16^{b}	6.72±0.93^{ab}	6.93±1.45^{ab}	6.72±1.18^{ab}	7.89±1.6^{a}

: male> female is significant (p<0.05)

APPENDIX D

PFIRRMANN CLASSIFICATION OF EACH PATIENT

Table D.1 - Pfirrmann Classification by patient.

	Pfirrmann Classification				
Code	**L1-L2**	**L2-L3**	**L3-L4**	**L4-L5**	**L5-S1**
1	III	IV	III	III	IV
2	I	I	II	II	IV
3	III	III	III	IV	V
4	III	III	IV	IV	IV
5	I	I	I	I	III
6	II	II	II	III	IV
7	II	II	I	I	III
8	II	II	II	III	II
9	III	III	II	II	II
10	II	III	II	II	IV
11	II	II	IV	III	IV
12	III	II	III	IV	III
13	II	III	II	III	III
14	IV	III	IV	IV	III
15	III	III	III	III	III
16	II	II	I	II	IV
17	III	II	III	III	IV
18	III	II	III	III	IV
19	IV	III	IV	IV	IV
20	II	II	II	II	IV
21	II	II	III	IV	V
22	II	I	I	I	III
23	II	II	II	III	IV
24	II	II	III	IV	II
25	III	II	III	III	III
26	II	III	III	IV	III