

RESEARCH

Open Access



CT-based surrogate parameters for MRI-based disc height and endplate degeneration in the lumbar spine

Thorsten Jentzsch^{1,2*}, Karin E. Mantel¹, Ksenija Slankamenac¹, Georg Osterhoff¹ and Clément M. L. Werner¹

Abstract

Purpose This study investigated potential use of computed tomography (CT)-based parameters in the lumbar spine as a surrogate for magnetic resonance imaging (MRI)-based findings.

Methods In this retrospective study, all individuals, who had a lumbar spine CT scan and MRI between 2006 and 2012 were reviewed ($n = 198$). Disc height (DH) and endplate degeneration (ED) were evaluated between Th12/L1-L5/S1. Statistics consisted of Spearman correlation and univariate/multivariable regression (adjusting for age and gender).

Results The mean CT-DH increased kranio-caudally (8.04 millimeters (mm) at T12/L1, 9.17 mm at L1/2, 10.59 mm at L2/3, 11.34 mm at L3/4, 11.42 mm at L4/5 and 10.47 mm at L5/S1). MRI-ED was observed in 58 (29%) individuals. CT-DH and MRI-DH had strong to very strong correlations (ρ 0.781-0.904, $p < .001$). MRI-DH showed higher absolute values than CT-DH (mean of 1.76 mm). There was a significant association between CT-DH and MRI-ED at L2/3 ($p = .006$), L3/4 ($p = .002$), L4/5 ($p < .001$) and L5/S1 ($p < .001$). A calculated cut-off point was set at 11 mm.

Conclusions In the lumbar spine, there is a correlation between disc height on CT and MRI. This can be useful in trauma and emergency cases, where CT is readily available in the lack of an MRI. In addition, in the middle and lower part of the lumbar spine, loss of disc height on CT scans is associated with more pronounced endplate degeneration on MRIs. If the disc height on CT scans is lower than 11 mm, endplate degeneration on MRIs is likely more pronounced.

Level and design Level III, a retrospective study.

Keywords Disc degeneration, Disc height, Endplate degeneration, MRI, CT

*Correspondence:

Thorsten Jentzsch
thorsten.jentzsch@balgrist.ch

¹Department of Traumatology, University Hospital Zürich, University of Zurich, Zurich, Switzerland

²Department of Orthopaedics, Balgrist University Hospital, Zurich, Switzerland



© The Author(s) 2024. **Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by-nc-nd/4.0/>.

Introduction

Disc degeneration (DD)

Intervertebral discs are located in the space between vertebral bodies and play an important role in movement, load distribution and spinal stability. They consist of a gelatinous nucleus pulposus and an annulus fibrosus [2]. Disc degeneration (DD) is subject to progressive alterations due to genetic predisposition [3], mechanics [4], and tissue changes [5]. This can manifest as disc height (DH) loss [6] and endplate degeneration (ED) [7]. Specifically, DD in the lumbar spine can be due to alterations of either the disc or the bone, each entailing different treatment options [8]. A concept of two phenotypes for DD has been suggested [9], indicating that they may be caused by alterations of the endplates of the vertebra or annulus of the disc. Endplate-driven DD has been associated with fractures and localization at the thoracic and upper lumbar spine [8]. Annulus-driven DD has been attributed to disc prolapse and localization at the lower lumbar spine [10].

Disc height (DH)

Disc height loss may result from reduced hydration capacity [11]. In turn, it leads to abnormal load forces in endplates making them more susceptible to injury [12]. Supero-inferior DH loss leads to outward bulging of redundant annulus fibrosus [13]. In the case of posterior bulging, neural structures may become compromised [14]. Disc height loss may also lead to a reduction of foraminal heights and spinal root compression [15]. This may ultimately result in spinal instability [16] and pain [17]. Limited studies have investigated the correlation of quantitatively measured DH on computed tomography (CT) scans and magnetic resonance imaging (MRI) [17–20].

Endplate degeneration (ED)

Endplate degeneration on MRIs was first described in detail by Modic et al. [21, 22] as three grades depending on signal intensity. It can evolve over time and may convert to a more severe grade [23]. It has been associated with low back pain [24]. Even though it seems to influence DD [7, 25], the exact pathomechanism remains unclear. Etiologic risk factors for ED have received little attention in research [18, 25, 26]. So far, mainly age [26] and disc changes such as disc herniation [25] and DH loss [18, 27] have been suggested as possible risk factors. However, DH has only been reported on a visual scale instead of providing exact quantitative measures [18].

Imaging

Several imaging techniques such as finite element models [28], macroscopy [29], conventional radiographs [30], and magnetic resonance imaging (MRIs) [31] have been

used in order to characterize DD. Up to now, the degree of DD has been compared on radiographs and MRIs [32]. Grading of ED is usually performed on MRIs. However, whether CT-based imaging of DH (CT-DH) might serve as a valuable alternative diagnostic tool for MRI-based ED (MRI-ED) remains underreported [18–20]. Recent studies have compared MRI-based synthetic CTs to conventional CTs, but using a special software to convert MRIs into CTs is not feasible in everyday clinical use [19, 20]. This may be especially important in trauma settings, where patients suffer from unstable fractures and surgeons may opt to increase DH when performing spondylodesis which may in turn influence ED and pain. Furthermore, anthropometric data on numeric measurements of CT-DH including the entire lumbar spine remain rare [34].

Clinical practice

In clinical practice, and especially in trauma settings, CT scans are much more readily available, faster and less problematic for claustrophobic patients than MRIs [33]. Usually, but depending on a hospital's infrastructure, CT scans can usually be obtained on the same day, but MRIs are usually not available until at least several days, often weeks later if there is no neurological emergency. In trauma settings, surgeries are therefore often performed without the availability of an MRI.

Aim

Therefore, our goal was to investigate the possible use of CT-based surrogate parameters for MRI-based findings in the lumbar spine in a typical clinical setting without using any additional software.

Methods

Individuals

We retrospectively identified all individuals, who presented to our hospital and underwent a CT scan and MRI of their lumbar spine, between 2006 and 2012 ($n=352$). Exclusion criteria involved fractures, osteomyelitis, metastasis, tumor or previous surgical intervention of the lumbar spine limiting the evaluation of DH or ED. These patients were excluded in order not to introduce any bias to the measurements by potentially non-degenerative changes. For example, any surgical intervention such as lumbar decompression surgery for disc herniation or fusion surgery for severe segment degeneration likely altered the anatomy of the DH and ED (e.g. in an attempt to achieve intercorporeal fusion) leading to substantial changes in the biology of these structures. The study has been approved by the local research ethics review committee (KEK-ZH Nr. 2011–0507). Methods were performed in accordance with relevant guidelines/regulations.

Radiology

A dual-source CT scanner (Somatom Definition, Siemens Healthcare, Forchheim, Germany) [35] and a 1.5-Tesla (T) MRI (Excite HDx, GE Healthcare, Waukesha, WI, USA) with a standard imaging protocol were used [36]. Sequences included sagittal T1-weighted (repetition time/echo time [TR/TE], 500/13 milliseconds [ms]; field of view [FOV], 240 mm), sagittal T2-weighted (TR/TE, 3160/112 ms; FOV, 240 mm), axial T2-weighted images (TR/TE, 3,160/112 ms; FOV, 160 mm), and sagittal short tau inversion-recovery images (STIR) (TR/TE, 4,760/44 ms; inversion time, 200 ms; FOV, 240 mm) (Fig. 1). Slice thickness was 3 millimeters (mm), spacing 1 mm, and number of excitations 2. Measurements were performed between the twelfth thoracic and the first sacral level on sagittal images. There was no inter-scanner variability due to the use of one CT scanner and one MRI. Quality assurance was guaranteed by regular maintenance at the institution where this study was performed (level 1 University trauma center) including adherence to calibration protocols.

Outline of measurements

Using patient charts was important in the evaluation process. Utilizing an Impax viewer (Agfa Health Care

GmbH, Bonn, Germany), one investigator carried out all of the measurements. Measurements were clearly defined and easy to measure, which is why only one investigator was chosen to perform all measurements. Reliability of measurements has been reported in other previous studies, but since intra- or interobserver correlation was not investigated in this report, a second investigator chose a random set of 10 patients to confirm the accuracy of the first investigator's measurements without finding any deviations in measurements [37, 38]. Disc height represents the distance between the upper and the lower endplate of two adjacent vertebrae. It was measured in the middle of the intervertebral disc in midsagittal planes in the entire lumbar spine between the twelfth thoracic (Th) level and first lumbar (L) level (Th12/L1) as well as the fifth lumbar and first sacral level (L5/S1) of CTs and MRIs according to previous reports (Fig. 2) [34]. Endplate degeneration was analyzed according to a grading scale introduced by Modic et al. (Fig. 3) [21, 22]. Grades were dependent on signal intensity on T1- and T2-weighted images. Grade 1 indicated accumulation of fibrovascular tissue in endplates with decreased signal intensity in T1-weighted images and increased signal intensity in T2-weighted images. Grade 2 indicated accumulation of lipid in endplates with increased signal intensity in



Fig. 1 Magnetic resonance imaging (T1- and T2-weighted) and computed tomography of the lumbar spine. Sagittal MRI (T1- [left] and T2-weighted [middle]) images as well as CT (right) image of the lumbar spine



Fig. 2 Disc height (DH) in the lumbar spine. Sagittal MRI (T1-weighted [left]) and CT (right) images with examples of DH measurements (millimeters [mm]) at L1/2 and L3/4

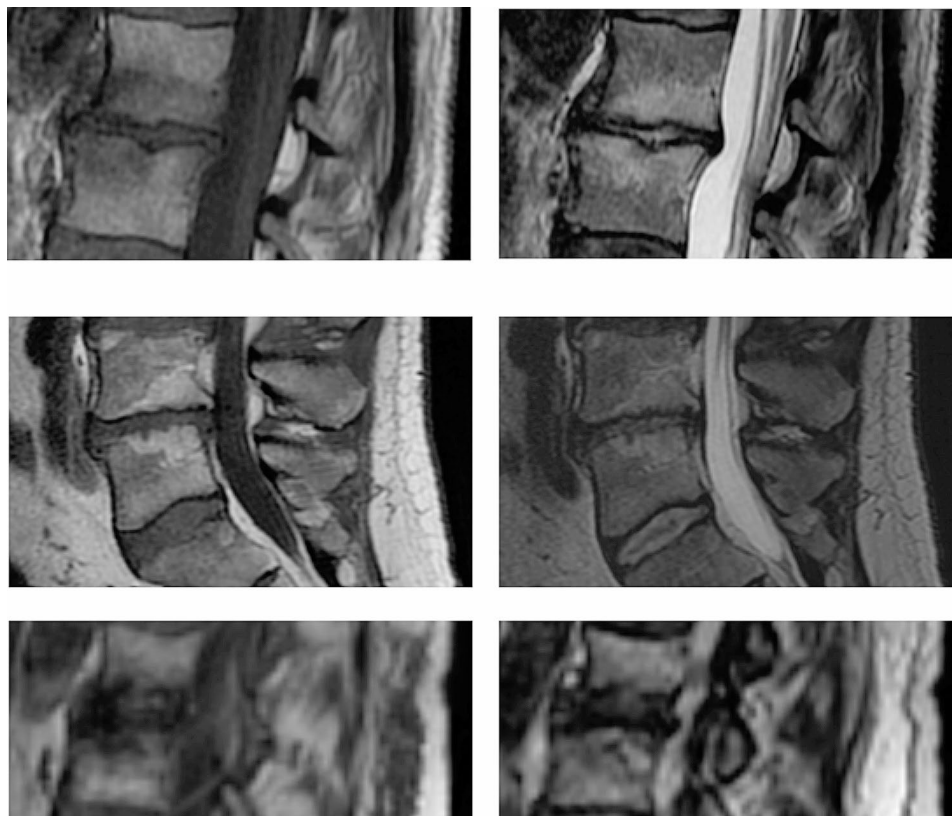


Fig. 3 Endplate degeneration (ED) in the lumbar spine. Sagittal MRI (T1- [left] and T2-weighted [right]) images of the lumbar spine. Grade 1 (top), grade 2 (middle) and grade 3 (bottom) of ED

Table 1 Disc height (DH) based on measurements (mean and standard deviation) of computed tomography (CT) and magnetic resonance imaging (MRI) according to lumbar level ($n = 198$)

Lumbar level	CT-DH	MRI-DH	Spearman's rho	P-value*
Th12/L1	8.04 (1.61)	9.61 (1.74)	0.781	< 0.001
L1/2	9.17 (1.86)	10.96 (2.05)	0.814	< 0.001
L2/3	10.59 (2.48)	12.43 (2.44)	0.860	< 0.001
L3/4	11.34 (2.59)	13.13 (2.87)	0.899	< 0.001
L4/5	11.42 (2.87)	13.08 (3.09)	0.904	< 0.001
L5/S1	10.47 (2.73)	12.37 (3.03)	0.863	< 0.001

*Spearman's rank correlation

T1- and T2-weighted images. Grade 3 indicated sclerosis of endplates with decreased signal intensity in T1- and T2-weighted images. Of note, we also reported the lumbar level that was affected by ED. The pelvic incidence was also recorded as previously described [39].

Pain

Patient charts were also evaluated for the presence of lumbar back pain.

Statistics

In a first step of the analysis, we expressed distribution of variables using means and standard deviation (SD) for normally distributed data, and medians and interquartile ranges for non-normally distributed data. We tested data for normality with the Kolmogorow-Smirnow test and performed quantile-quantile plots of dependent variables.

We performed a simple linear or logistic regression (without adjustment for confounders), and in the main analysis, a multivariable linear or logistic regression model adjusted for potential confounders, such as age and sex [6, 8]. For all results, we reported point estimates, 95% (%) confidence intervals (CI) and p -values (≤ 0.05 considered significant). Spearman's rank correlation (Spearman's rho) was used to compare CT- and MRI-based measurements. As a guideline, values of $> 0.6 - \leq 0.8$ indicate strong correlation and values between $> 0.8 - \leq 1.0$ indicate very strong correlation. A receiver operating curve (ROC) analysis was performed to calculate a cut-off point for the association between CT-DH and MRI-ED. It needs to be taken into account that this has several drawbacks, such as arbitrary selection, but it was carefully chosen. We performed the statistical analyses using the statistical program Stata (Version 12; Stata Corporation, College Station, Texas, United States of America).

Table 2 Endplate degeneration (ED) according to lumbar levels ($n = 196$)

	ED		
	Grade 1	Grade 2	Grade 3
Th12/L1-L3/4	5 (55.6%)	10 (23.3%)	3 (50%)
L4/5-L5/S1	4 (44.4%)	33 (76.7%)	3 (50%)

Results

Individuals

Our study included 198 individuals. The mean age was 50 (range 18–90) years. There were 75 (38%) females and 123 (62%) males. One hundred and fifty-five individuals were excluded according to our exclusion criteria.

Disc height (DH)

Our results for CT-DH and MRI-DH (Fig. 2) according to its specific lumbar level are shown in detail in Table 1. There was a significant ($p < .001$) craniocaudal increase from Th12/L1 to L5/S1. CT-DH was significantly ($p < .001$) correlated with MRI-DH at the entire lumbar spine. Slightly higher values were found on MRI measurements. The mean difference between CT-DH and MRI-DH for the investigated levels was 1.76 mm (1.57 mm for Th12/L1, 1.79 mm for L1/2, 1.84 mm for L2/3, 1.79 mm for L3/4, 1.66 mm for L4/5 and 1.90 mm for L5/S1).

Endplate degeneration (ED)

Endplate degeneration (Fig. 3) was absent (grade 0) in 138 (70%) individuals. It was present in 58 (29%) individuals, whereof grade 1 was seen in nine (5%) individuals, grade 2 in 43 (21%) individuals and grade 3 in six (3%) individuals. It could not be clearly determined in two (1%) individuals. Endplate degeneration according to its lumbar level is shown in Table 2.

The mean PI did not differ between individuals without and with endplate degeneration (mean PI 56.4 [SD 9.6] versus 57.8 [10.5] degrees, adjusted differences -0.11 [95% CI $-3.68 - 3.46$, $p = .953$]).

Individuals with ED also were more likely to have lumbar back pain, irrespective of age and sex (adjusted odds ratio 2.52 [95% CI 1.15–5.53, $p = .021$; when comparing ED grade 2 vs. 0 [grades 1 and 3 were not included in the regression analysis due to small sample sizes]).

Disc height (DH) and endplate degeneration (ED)

A significant association was shown between CT-DH and MRI-ED at the middle and lower lumbar spine with a cut-off point at 11 mm. This indicates that DH values < 11 mm are significantly ($p = .017$) associated with present ED compared to DH values ≥ 11 mm, which are associated with absent ED. Statistical associations did not change when adjusting for age and sex (Table 3). Disc

Table 3 Disc height (DH) (mean [standard deviation]) according to endplate degeneration (ED) at specific lumbar levels ($n = 196$)

Level	DH		Unadjusted differences (95% CI, p -value [*])	Adjusted differences (95% CI, p -value [*])
	ED grade 0 ($n = 138$)	ED grade ≥ 1 ($n = 58$)		
Th12/L1	9.5 (1.6)	9.8 (2.0)	0.23 (-0.31–0.77, $p = .395$)	-0.24 (-0.80–0.32, $p = .400$)
L1/L2	10.9 (1.8)	11.0 (2.5)	0.06 (-0.57–0.69, $p = .854$)	-0.14 (-0.82–0.54, $p = .676$)
L2/L3	12.8 (2.1)	11.6 (2.9)	-1.19 (-1.92 – -0.45, $p = .002$)	-1.10 (-1.89–0.31, $p = .006$)
L3/L4	13.6 (2.4)	11.9 (3.3)	-1.62 (-2.47 – -0.78, $p < .001$)	-1.47 (-2.37–0.56, $p = .002$)
L4/L5	13.7 (2.5)	11.4 (3.7)	-2.30 (-3.19 – -1.40, $p < .001$)	-1.99 (-2.96 – -1.03, $p < .001$)
L5/S1	13.0 (2.4)	10.9 (3.8)	-2.05 (-2.95 – -1.15, $p < .001$)	-2.04 (-3.03 – -1.05, $p < .001$)

^{*}Wald test

Abbreviation: confidence interval (CI)

height and ED were not significantly associated at the thoracolumbar junction or upper lumbar spine.

Discussion

Our study reports several novel findings regarding the ongoing search for objective markers for ED. We found CT-DH to be significantly correlated with MRI-DH at all levels of the lumbar spine. Furthermore, we showed that CT-DH loss and increased MRI-ED are associated at the middle and lower lumbar spine, irrespective of adjustment for age and sex.

Disc height (DH) on computed tomography (CT) and magnetic resonance imaging (MRI)

Our study evaluates the association between measurements of DH on CTs compared to MRIs. We also provide important anthropometric data on numeric CT-measurements of DH including the entire lumbar spine. There are significant correlations between CT-DH and MRI-DH. Measurements of MRI-DH were mildly higher than CT-DH, which may be due to better visualization of the disc and clearer borders of cartilage.

Taking our findings for the association of DH with ED into account, we suggest decreased CT-DH as a surrogate marker for a more severe MRI-ED in the lumbar spine. This may be valuable to clinicians because CT scans are much more common and readily available, especially in trauma settings and preoperative planning, where MRI is not indicated or available [33]. For example, a poly-traumatized patient with a lumbar vertebral fracture in need of spondylodesis showing a decreased DH on a CT scan may benefit from an increase in DH during reduction in order to possibly halt ED and pain. However, clinical decision-making should be based on comprehensive assessments and clinicians should only rely on stand-alone CT scans when indicated. For example, if a patient is suspected to have a disc herniation in addition to a fracture, or if spinal fusion surgery is considered for degenerative disease, an additional MRI is warranted. Further prospective studies would be needed before implementing this into routine clinical practice. Furthermore, younger patients with chronic pain may only have

to undergo an MRI for assessment of their degenerative changes without exposing them to radiation from CT scans.

Numeric DH measurements have mostly been carried out on conventional radiographs [30], which may be too inaccurate for specific conclusions [34]. In order to provide researchers and clinicians with valuable information about this issue, Zhou et al. [34] opted to provide a database of lower lumbar spinal characteristics based on CTs of 126 patients with low back pain. Their measuring technique resembled ours and mean DH in the lower lumbar segments were 11.6 (± 1.8) mm for L3/4, 11.3 (± 2.1) mm for L4/5 and 10.7 (± 2.1) mm for L5/S1. The decrease in DH from L4/5 to L5/S1 is similar to our findings and may be due to increased axial load at L5/S1. Another study by Jaovisidha et al. [32] compared X-rays and MRIs in 100 patients with back pain. They reported that DH loss was associated with DD, that is, anterior DH < 11.3 mm and posterior DH < 5.5 mm at L5/S1. The anterior cut-off is close to ours, which was set at the center of the vertebrae.

This adds valuable clinical information to the current literature, where a recent study by Morbée et al. of 30 participants found equivalency of MRI-based synthetic CTs to conventional CTs [19]. Another study by Schwaiger et al. studied 104 patients with fractures and degenerative changes and also found that morphologic assessment of bone pathologies was feasible with MRI-based synthetic CTs to conventional CTs [20]. Disc height was measured, however, the exact location in the lumbar spine was not stated. It showed excellent intraclass correlation of 0.99. However, in every day clinical practice, using a special software to convert MRIs into CTs is not feasible.

Disc height (DH) and endplate degeneration (ED)

As suggested by our findings, CT-DH loss may lead to increased MRI-ED in the middle and lower lumbar spine. The thoracolumbar junction and upper lumbar spine remain largely unaffected. The explanation may be found in the fact that reduced DH in the middle and lower lumbar spine leads to increased abnormal load forces in the endplates making them more susceptible to injury [12].

This is consistent with previous reports. In a prospective, longitudinal study of MRIs in patients with chronic low-back pain, Kertulla et al. [18] evaluated degenerative changes in the lumbar spine including DH with regard to ED. They concluded that MRI-DH loss was associated with increased MRI-ED. However, they did not measure DH quantitatively, but only reported a visual grading scale that may have been more subjective. Their visual grading scale involved comparison of DH at the affected level to the level above. They also found DH loss and increased ED to be located at the lower lumbar spine, namely at L4/5 and L5/S1. Furthermore, a study by Iguuchi et al. [6] pointed out that DH loss and older age were the most common parameter for DD and spinal instability. However, their measurement of DH was rather complicated and may be too time consuming for easy clinical use. Besides, they only examined one segment, L4/5, and not the entire lumbar spine.

It is interesting to search for explanations why the association of DH loss and increased ED are dependent on spinal levels. Normally, thoracolumbar DH, mainly involving the annulus [40] and disc volume, mainly involving the nucleus [41], increase in a craniocaudal fashion. This is supposed to reduce the risk of DD, such as disc prolapse in the lower lumbar spine due to larger vertical deformations and smaller pressure descents. This craniocaudal change does not seem to be accompanied by changes in endplates because the increase in endplate area is much less pronounced than the decrease in DD [42]. Compared to lumbar levels [8], decreases in thoracic DH represent a relatively low risk for DD, such as disc prolapse [43] and back pain [44]. Besides, thoracic ED is more uncommon than lumbar ED. Therefore, the ratio of DH and endplates is smaller in the lumbar spine. If there is DH loss due to increased axial loading at the lower lumbar spine [10], endplates may be more prone to ED.

The cut-off value for increased risk of ED with $DH < 11$ mm can be used to educate patients about their risk of ED, and, although not the topic of this manuscript, potential back pain, if their DH falls below the threshold of 11 mm. If a patient has a DH well beyond 11 mm, patients may rest assured and may not be followed up as closely. However, if DH nears 11 mm, a closer follow-up time period may be chosen. Additionally, MRI scans may be ordered more liberally if DH nears 11 mm to search for ED.

Pelvic incidence

We did not find an association between PI and ED. While PI is associated with facet joint arthritis [39], pelvic anatomy may not influence endplates as much due to less influence on the axial pressure in the anterior than the posterior spine. It therefore appears that static spinal

alignment may be less of an influencing factor for ED than other multifactorial risk factors (e.g. genetics, aging, metabolic factors).

Pain

Our study also showed that endplate degeneration at the lumbar spine was associated with low back pain. Similarly, an increase in angiogenesis and sensory nerve endings in endplates along with ED of low back pain patients was found by Brown et al. [45]. Furthermore, in a retrospective study of 150 elderly patients with chronic low back pain, Ma et al. [46] reported a high incidence of ED as well. There are several potential explanations regarding the relationship between DH, ED and low back pain. Endplates have free nerve endings and irritation (e.g. by inflammatory response of mechanical instability) of these nerves can result in pain. This is relevant for all patients with lumbar back pain, as endplate degeneration may be treated with lumbar fusion surgery, potentially alleviating low back pain.

Limitations

The retrospective nature of this study is a limiting factor because of a potential bias of participant selection. The generalizability to other populations, healthcare settings, and timeframes needs to be done with caution. Our results mainly correspond to the local population of patients presenting to the emergency department with a reason non-related to the spine, but this patient population may be a particular subset of patients with potential different prevalence of back pain than the general population. All of our participants presented to a hospital instead of being randomly recruited. Thus, they may have been more ill and may not represent the general population. If this was the case, findings may have been influenced in a way that DH was lower and ED degeneration more common than in a normal population. Furthermore, patients with pathologies potentially affecting the discs were excluded with the potential for selection bias and findings of this study cannot be inferred to these populations. For example, fractures were excluded as these may also have ruptured discs leading to changes in disc morphology, such as reduced DH without ED. Nonetheless, we tried to exclude individuals with a morbidity that could have influenced our measurements. Besides, it may be difficult to acquire healthy individuals for a study like ours, because CT scanning is associated with a considerable dose of radiation and healthy individuals may be hesitant to undergo unnecessary CT scans. There were several subgroup-analyses, which may render some findings underpowered, so findings need to be interpreted with care. However, a sample size of 198 patients is rather large, and a post hoc power analysis for the association between DH and endplate degeneration yielded 97%. In

addition, the quality and completeness of the CT and MRI data was thoroughly assessed to ensure their reliability. Furthermore, although this study identifies correlations between CT-based parameters and MRI-based findings, it does not establish causation.

Implementing CT-based DH measurements as a routine diagnostic tool may provide the benefits of high-resolution imaging for objective measurements, diagnostics, and potentially surgical planning, but is associated with radiation exposure (usually around 6 millisieverts, which is equal to around 2 years of background radiation) and costs (several hundred dollars), which are higher than in X-rays or MRI. Therefore, CTs should only be used when deemed necessary. Since the mean age of patients in this study was 50 years, clinicians may be particularly careful in applying the findings of this study to younger patients.

There was no control group or healthy individuals for comparison in this study. Even so, we were able to include a large number of individuals in order to provide a sufficient sample size in each group for adequate statistical analysis. Besides, we were able to adjust for age and sex because increasing age leads to stiffness of the annulus and female sex is associated with increased bone fragility [6, 8]. Although age and gender was adjusted for, there are several other potential confounders, such as comorbidities, patient history, and other clinical variables that could influence the findings of this study. The reported cut-off point at the middle and lumbar spine may not be applicable to different lumbar levels because there is a general cranio-caudal trend toward lower DH and further studies are recommended. Furthermore, in vivo measurements on MRI deliver precise information on DH and ED in not only specific levels, but in the the entire lumbar spine when compared to previous cadaveric studies [8]. There were no unexpected limitations encountered during data collection of analysis.

The findings should be validated in an independent dataset or through prospective studies to assess the generalizability of the results. Future studies may opt to utilize U-Net models for automatic segmentation, within-modality synthesis, image-based radiomics and machine learning systems including clinical data, which may take into account the patient-reported outcome measures before and after surgical treatment, such as spinal fusion, to increase the predictive performance of DH for endplate degeneration as previously shown in other fields, such as cognitive decline in Parkinson's disease using clinical and computed tomography [47–51].

Conclusions

In the lumbar spine, there is a correlation between disc height on CT and MRI. This can be useful in trauma and emergency cases, where CT is readily available in the lack of an MRI. In addition, in the middle and lower

part of the lumbar spine, loss of disc height on CT scans is associated with more pronounced endplate degeneration on MRIs. If the disc height on CT scans is lower than 11 mm, endplate degeneration on MRIs is likely more pronounced. Therefore, if a patient presents with an initial CT scan with a disc height close to or lower than 11 mm, physicians need to be more liberal in ordering an MRI to assess for endplate degeneration, which may cause pain and may be treated with spinal fusion.

Acknowledgements

An altered version of the abstract was presented as a poster presentation at the annual conference of the German Spine Society (DWG), Leipzig, Germany, December 11–13, 2014. The abstract was also presented as a poster presentation at the annual conference of the swiss orthopaedics, Montreux, Switzerland, June 06–08, 2018.

Author contributions

TJ: idea, conception and design, analysis and interpretation of data, drafting the manuscript. KEM: acquisition of data. KS: analysis of data. GO: conception and design. CML: supervision of the project. All: revision of the manuscript, final approval of the version to be published.

Funding

None.

Data availability

All data analysed during this study are included in this article.

Declarations

Ethics approval and consent to participate

Ethical approval was obtained prior to the start of the study (Ethics Committee Zurich, KEK-ZH Nr. 2011–0507). According to the local ethics committee, informed consent to participate was waived (Ethics Committee Zurich, KEK-ZH Nr. 2011–0507) due to the retrospective nature of this study.

Consent to publish

Not applicable.

Competing interests

The authors declare no competing interests.

Received: 6 August 2023 / Accepted: 6 August 2024

Published online: 13 August 2024

References

1. Jeong JG et al. Biomechanical Effect of Disc Height on the Components of the Lumbar Column at the Same Axial Load: A Finite-Element Study. *J Healthc Eng.* 2022;2022:7069448.
2. Mohd Isa IL et al. Discogenic low back Pain: anatomy, pathophysiology and treatments of intervertebral disc degeneration. *Int J Mol Sci.* 2022. 24(1).
3. Battié MC, et al. Genetic and environmental effects on disc degeneration by phenotype and spinal level: a multivariate twin study. *Spine (Phila Pa 1976).* 2008;33(25):2801–8.
4. Adams MA, Dolan P. *Spine Biomech J Biomech.* 2005;38(10):1972–83.
5. Adams MA, Roughley PJ. What is intervertebral disc degeneration, and what causes it? *Spine (Phila Pa 1976).* 2006;31(18):2151–61.
6. Iguchi T, et al. Intimate relationship between instability and degenerative signs at L4/5 segment examined by flexion-extension radiography. *Eur Spine J.* 2011;20(8):1349–54.
7. Kuisma M, et al. A three-year follow-up of lumbar spine endplate (Modic) changes. *Spine (Phila Pa 1976).* 2006;31(15):1714–8.
8. Dolan P, et al. Intervertebral disc decompression following endplate damage: implications for disc degeneration depend on spinal level and age. *Spine (Phila Pa 1976).* 2013;38(17):1473–81.

9. Adams MA, Dolan P. Intervertebral disc degeneration: evidence for two distinct phenotypes. *J Anat.* 2012;221(6):497–506.
10. Bazrgari B, Shirazi-Adl A, Arjmand N. Analysis of squat and stoop dynamic liftings: muscle forces and internal spinal loads. *Eur Spine J.* 2007;16(5):687–99.
11. Urits I, et al. Stem cell therapies for treatment of Discogenic Low Back Pain: a Comprehensive Review. *Curr Pain Headache Rep.* 2019;23(9):65.
12. Adams MA, et al. Mechanical initiation of intervertebral disc degeneration. *Spine (Phila Pa 1976).* 2000;25(13):1625–36.
13. Jinkins JR. Acquired degenerative changes of the intervertebral segments at and suprajacent to the lumbosacral junction. A radioanatomic analysis of the nondiscal structures of the spinal column and perispinal soft tissues. *Eur J Radiol.* 2004;50(2):134–58.
14. Resnick D. Degenerative diseases of the vertebral column. *Radiology.* 1985;156(1):3–14.
15. Kuslich SD, Ulstrom CL, Michael CJ. The tissue origin of low back pain and sciatica: a report of pain response to tissue stimulation during operations on the lumbar spine using local anesthesia. *Orthop Clin North Am.* 1991;22(2):181–7.
16. Zhao F, et al. Discogenic origins of spinal instability. *Spine (Phila Pa 1976).* 2005;30(23):2621–30.
17. Videman T, et al. Associations between back pain history and lumbar MRI findings. *Spine (Phila Pa 1976).* 2003;28(6):582–8.
18. Kerttula L, et al. Modic type I change may predict rapid progressive, deforming disc degeneration: a prospective 1-year follow-up study. *Eur Spine J.* 2012;21(6):1135–42.
19. Morbée L, et al. MRI-based synthetic CT of the lumbar spine: geometric measurements for surgery planning in comparison with CT. *Eur J Radiol.* 2021;144:109999.
20. Schwaiger BJ, et al. CT-like images based on T1 spoiled gradient-echo and ultra-short echo time MRI sequences for the assessment of vertebral fractures and degenerative bone changes of the spine. *Eur Radiol.* 2021;31(7):4680–9.
21. Modic MT, et al. Magnetic resonance imaging of intervertebral disk disease. Clinical and pulse sequence considerations. *Radiology.* 1984;152(1):103–11.
22. Modic MT, et al. Degenerative disk disease: assessment of changes in vertebral body marrow with MR imaging. *Radiology.* 1988;166(1 Pt 1):193–9.
23. Luoma K, et al. MRI follow-up of subchondral signal abnormalities in a selected group of chronic low back pain patients. *Eur Spine J.* 2008;17(10):1300–8.
24. Suryadevara M, et al. Role of end plate changes and Paraspinal Muscle Pathology in Lower Back Pain: a narrative review. *Cureus.* 2024;16(5):e61319.
25. Jensen TS, et al. Predictors of new vertebral endplate signal (Modic) changes in the general population. *Eur Spine J.* 2010;19(1):129–35.
26. Boos N, et al. Classification of age-related changes in lumbar intervertebral discs: 2002 Volvo Award in basic science. *Spine (Phila Pa 1976).* 2002;27(23):2631–44.
27. Rohlmann A, et al. Analysis of the influence of disc degeneration on the mechanical behaviour of a lumbar motion segment using the finite element method. *J Biomech.* 2006;39(13):2484–90.
28. Nispel K et al. Recent advances in coupled MBS and FEM models of the Spine—A review. *Bioeng (Basel).* 2023. 10(3).
29. NACHEMSON A. Lumbar intradiscal pressure. Experimental studies on post-mortem material. *Acta Orthop Scand Suppl.* 1960;43:1–104.
30. Saraste H, et al. Radiographic measurement of the lumbar spine. A clinical and experimental study in man. *Spine (Phila Pa 1976).* 1985;10(3):236–41.
31. Abel F, et al. Imaging of Discogenic and Vertebrogenic Pain. *Radiol Clin North Am.* 2024;62(2):217–28.
32. Jaovisidha S, et al. Degenerative disk disease at lumbosacral junction: plain film findings and related MRI abnormalities. *J Med Assoc Thai.* 2000;83(8):865–71.
33. van Rijn JC, et al. Observer variation in the evaluation of lumbar herniated discs and root compression: spiral CT compared with MRI. *Br J Radiol.* 2006;79(941):372–7.
34. Zhou SH, et al. Geometrical dimensions of the lower lumbar vertebrae—analysis of data from digitised CT images. *Eur Spine J.* 2000;9(3):242–8.
35. Jentzsch T, et al. Hyperlordosis is Associated with Facet Joint Pathology at the lower lumbar spine. *J Spinal Disord Tech;* 2013.
36. Winklhofer S, et al. Magnetic resonance imaging frequently changes classification of acute traumatic thoracolumbar spine injuries. *Skeletal Radiol.* 2013;42(6):779–86.
37. Udby PM et al. The clinical significance of the Modic Changes Grading score. *Global Spine J.* 2022; p. 21925682221123012.
38. Chen XL, et al. Relation of lumbar intervertebral disc height and severity of disc degeneration based on Pfirrmann scores. *Heliyon.* 2023;9(10):e20764.
39. Jentzsch T, et al. Increased pelvic incidence may lead to arthritis and sagittal orientation of the facet joints at the lower lumbar spine. *BMC Med Imaging.* 2013;13:34.
40. Koeller W, Meier W, Hartmann F. Biomechanical properties of human intervertebral discs subjected to axial dynamic compression. A comparison of lumbar and thoracic discs. *Spine (Phila Pa 1976).* 1984;9(7):725–33.
41. Brinckmann P, Biggemann M, Hilweg D. Prediction of the compressive strength of human lumbar vertebrae. *Spine (Phila Pa 1976).* 1989;14(6):606–10.
42. Panjabi MM, et al. Human lumbar vertebrae. Quantitative three-dimensional anatomy. *Spine (Phila Pa 1976).* 1992;17(3):299–306.
43. Niemeläinen R, et al. The prevalence and characteristics of thoracic magnetic resonance imaging findings in men. *Spine (Phila Pa 1976).* 2008;33(23):2552–9.
44. Niemeläinen R, Battié MC, Videman T. Risk indicators for severe upper or mid back pain in men. *Spine (Phila Pa 1976).* 2011;36(5):E326–33.
45. Brown MF, et al. Sensory and sympathetic innervation of the vertebral endplate in patients with degenerative disc disease. *J Bone Joint Surg Br.* 1997;79(1):147–53.
46. Ma Z, et al. [The study on the relationship between modic change and disc height together with lumbar hyperosteoecy]. *Zhonghua Wai Ke Za Zhi.* 2013;51(7):610–4.
47. Rezaeijoo SM, et al. Segmentation of the prostate, its zones, anterior fibromuscular stroma, and urethra on the MRIs and multimodality image fusion using U-Net model. *Quant Imaging Med Surg.* 2022;12(10):4786–804.
48. Rezaeijoo SM et al. Within-modality synthesis and Novel Radiomic evaluation of Brain MRI scans. *Cancers (Basel).* 2023. 15(14).
49. Lam LHT, et al. Molecular subtype classification of low-grade gliomas using magnetic resonance imaging-based radiomics and machine learning. *NMR Biomed.* 2022;35(11):e4792.
50. Le VH, et al. Development and validation of CT-Based Radiomics Signature for overall survival prediction in multi-organ Cancer. *J Digit Imaging.* 2023;36(3):911–22.
51. Hosseinzadeh M et al. Prediction of Cognitive Decline in Parkinson's Disease Using Clinical and DAT SPECT Imaging Features, and Hybrid Machine Learning Systems. *Diagnostics (Basel).* 2023;13(10).

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.