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# Frequency of Hypoplasia of the Vertebral Body at L5, and Its Relationship with Degeneration in Patients with Low Back Pain

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## ABSTRACT

**AIM:** To explain the association between vertebral body hypoplasia and degenerative changes in the disco-vertebral complex and facet joints, and to assess the incidence of hypoplasia of the vertebral body at the L5 level.

**MATERIAL and METHODS:** A retrospective analysis was made of 3,100 patients aged 20–50 years who underwent lumbar MRI with a complaint of back pain, of which 55 were identified with vertebral body hypoplasia. Intervertebral disc degeneration was evaluated in the study using the Pfirrmann and Modified Pfirrmann classification systems, while degenerative changes in the vertebrae endplate were assessed using the Modic classification system. Osteoarthritis of the facet joint was graded at the L4–5 level, and spondylolysis and spondylolisthesis rates were compared between the control group and the hypoplasic group.

**RESULTS:** The incidence of hypoplasia of the vertebral body at the L5 level was found to be 1.8% in the population with back pain in the 20–50-year age group. In the hypoplasia group, disc degeneration was detected at a higher rate than in the control group ($p<0.001$). The distribution of Modic signal changes in the superior and inferior endplates of the vertebrae differed significantly between the hypoplasia and control groups ($p<0.001$).The rate of spondylolysis was 7.7% in the control group and 65.5% in the hypoplasia group ($p<0.001$), and spondylolisthesis was significantly more common in the hypoplasia group (18.4%, $p<0.001$). In addition, facet joint degeneration was identified more frequently in the hypoplasia group. Degenerative findings were detected in 74.5% of the right posterior intervertebral joints, and in 70.9% of the left posterior intervertebral joints in the hypoplasia group.

**CONCLUSION:** Vertebral body hypoplasia is a predisposing factor for disc degeneration, facet osteoarthritis and degeneration in the vertebral endplates, and has also been associated with spondylolysis and spondylolisthesis.

**KEYWORDS:** Spondylolysis, Spondylolisthesis, Vertebral body hypoplasia, Spinal degenerative disease, Wedge shape vertebra

## INTRODUCTION

Many anatomical and radiological studies havereported a progressive increase in the vertebral corpus dimensions from the C1 vertebra to the L5 vertebra level (3). Hypoplasia refers to cases in which the anterior-posterior (A-P) diameter in the upper vertebra is greater than the A-P diameter in the adjacent lower vertebra (15). In the late stages of chondrification and ossification, growth abnormalities in the centre of vertebral bodies can cause hypoplasia, most commonly at the L5 vertebral level (4).

Spondylolisthesis has been shown to frequently accompany hypoplasia of the vertebral body at L5 (30). Accordingly, in the present study we determine whether vertebral body hypoplasia observed at the L5 level is a predisposing factor for spinal degenerative disease through a comparison of a patient group with a randomized control group without hypoplasia,

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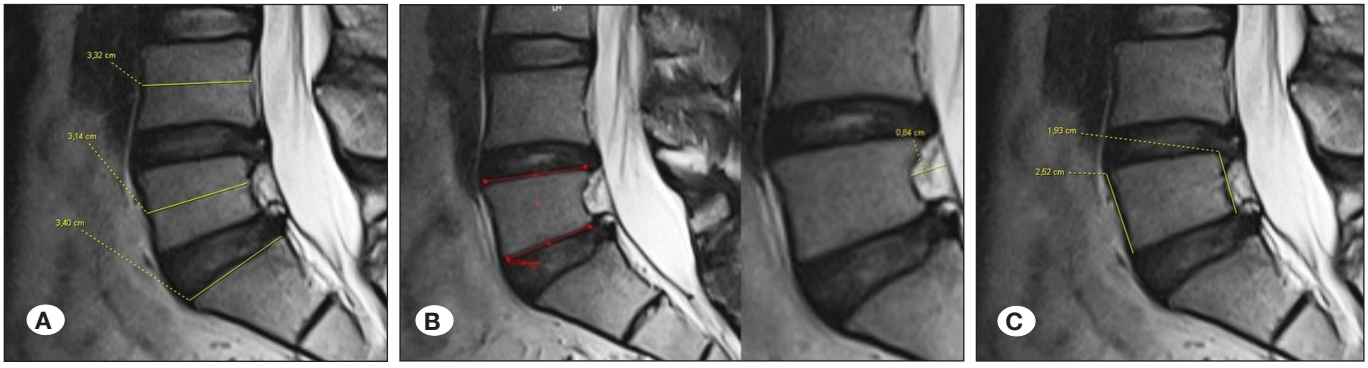


**Figure 1: A)** T2-weighted sagittal plane MR image, L4, L5 and S1 vertebra anteroposterior diameter measurements. **B)** T2-weighted sagittal plane MR image, L5 vertebra wedging angle measurement, L5 vertebra prevertebral fat tissue thickness measurement. **C)** T2-weighted sagittal plane MR image, L5 vertebra anterior and posterior edge height measurements.

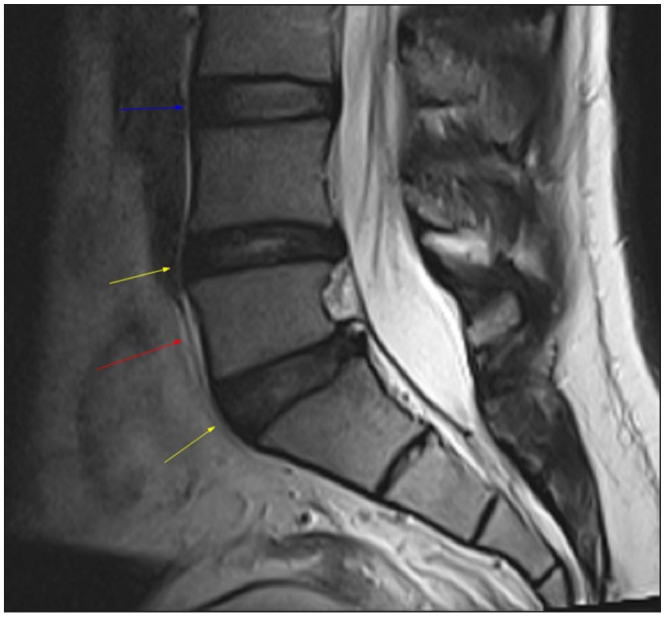

**Figure 2:** T2-weighted sagittal plane MR image, blue arrow: Normal disc, yellow arrow: Degenerate disc, red arrow: Hypoplastic vertebra.

making use of systems that classify lumbar degenerative disease based on MRI findings. In addition, we compare the frequency of spondylolysis and spondylolisthesis in cases of vertebral body hypoplasia with that of a control group, and discuss the possible reasons for the differences.

## MATERIAL and METHODS

A total of 3,100 patients who presented to our clinic with lower back pain between August 2016 and May 2018, and who underwent a lumbar MRI in a 1.5T MAGNETOM Aeradevice (Siemens Healthineers, Erlangen, Germany), a 1.5T GyroscanIntera (Philips, Best, The Netherlands) device and a 1.5T Achieva (Philips, Best, The Netherlands) device, were evaluated retrospectively from picture archiving and a communication system (PACS). As the standard protocol for a lumbar MRI examination, 4 mm-thick T2-weighted fast spin-echo (FSE) (time to repetition (TR)/time to echo (TE) 3,300 ms/91 ms) sequences parallel to the discs between the L1–S1 vertebrae in the axial plane, and 4 mm-thick T1-weighted FSE (TR/TE 440 ms/15 ms) and T2-weighted FSE (TR/TE 3,600 ms/105 ms) sequences in the sagittal plane were evaluated.

A total of 55 L5 vertebral body hypoplasia cases, including 33 males and 22 females, with a mean age of 37.2 (20–50) years were identified. The control group included 300 people (163 male, 137 female) without corpus hypoplasia and with a mean age of 37 (20–50), who were selected through a random sampling method from among 3,045 people with similar demographic characteristics. People aged below 20 or over 50 years, and those with hyperparathyroidism, hypothyroidism, Cushing's disease, diabetes mellitus, renal osteodystrophy, rheumatologic disease, infection, tumoral lesions, scalloping masses, connective tissue disease, glucocorticoid, bisphosphonate or antirheumatoid drug use, tropism in the posterior intervertebral joint and lumbosacral transitional vertebrae were excluded from the control group (3,14,15).

The hypoplasia examination involved the measurement of AP diameter from the middle section for the L4 and L5 vertebral bodies, and from the midsagittal line at the superior endplate level for S1 (31) (Figure 1A-C). Cases with a minimum diameter difference of 2 mm between the L4–L5 vertebral bodies were included in the hypoplasia group (30). For the evaluation of lumbar degenerative disease, changes at the level of the L5 vertebra, the adjacent discs and the posterior intervertebral joint were examined, and the spondylolysis-spondylolisthesis rates were compared.

The modified Pfirrmann system was used for the classification of disc degeneration, in which grade 1 refers to the absence of disc degeneration and grade 8 indicates disc collapse (9) (Figure 2, Table I). Degeneration in the vertebral endplates, on the other hand, was assessed using the classification system described by Modic et al., which is based on hypointense and hyperintense oedema in T1-weighted and T2-weighted images, respectively, hyperintense fatty changes in T1- and T2-weighted images, and a hypointense appearance, indicating sclerosis in T1- and T2-weighted images (17,25).

**Table I:** Comparison of the L5 Wedging Angle, L5 Anterior and Posterior Height and Fat Tissue Thickness (mm) Between the Study Groups

| | Hypoplasia Group (n=55) | | Control Group (n=300) | | *p |
|---|---|---|---|---|---|
| | Median | Min-Max | Median | Min-Max | |
| **L5 wedging angle** | 14 | 8-20 | 8 | 0-18 | **<0.001** |
| **L5 anterior height** | 28.0 | 22.2-31.5 | 27.8 | 20.0-34.9 | 0.768 |
| **L5 posterior height** | 19.9 | 15.2-25.3 | 24.0 | 18.0-31.1 | **<0.001** |
| **Fat tissue thickness** | 6.0 | 2.0-9.0 | 2.0 | 1.0-6.0 | **<0.001** |

**Mann-Whitney U test;* ***Min:*** *Minimum,* ***Max:*** *Maximum.*

Spondylolysis was defined as the presence of a unilateral or bilateral pars interarticularis defect in the absence of a shift in the vertebral body, while spondylolisthesis was evaluated as a slipping of the vertebra together with the upper vertebral column over the inferior vertebral body.

### Statistical Analysis

The measurements were presented as mean and standard deviation (SD), or as median (median) or minimum/maximum values for numerical data. Kolmogorov-Smirnov and Shapiro-Wilk tests were used to determine whether the numerical values obtained from the measurement were compatible with a normal distribution. For the comparison of numerical values between paired groups, an Independent-samples t-test (Student's t-test) was used for the normally distributed data, and a Mann-Whitney U test was conducted for data that did not comply with a normal distribution. Nominal or ordinal data obtained through counting were presented as numbers and percentages. A Chi-square test was conducted for the comparison of nominal or ordinal data between independent groups. The statistical analyses were performed using IBM SPSS Statistics for Windows (Version 25.0. Armonk, NY: IBM Corp.). A P value below 0.05 was considered statistically significant.

## RESULTS

Of the total 3,100 cases who presented to our hospital with lower back pain and who underwent an MRI were examined, hypoplasia of the L5 vertebral body was detected in 55. The frequency of this anomaly among the asymptomatic cases in the 20–50 years group was 1.8%.

Subsequently, 55 patients identified with vertebral body hypoplasia (33 males, 22 females) and 300 controls (163 males and 137 females) were included in the sample. There was no statistically significant difference between the hypoplasia and control groups in terms of the male/female ratio (1.5 versus 1.2; 0.782),or mean age (37.2 ± 8.7 versus 37.0 ± 8.8 years; p=0.956).

No significant difference was found between the two groups in terms of the L4/AP and S1/AP diameters (p=0.495 and p=0.973, respectively). The L5 AP diameter was significantly lower in the hypoplasia group (29.8 ± 2.2 mm) than in the control group (32.1 ± 3.0 mm) (p<0.001).

Statistically significant differences were noted between the hypoplasia and control groups in the absolute values of the L4–L5 and L5–S1 AP diameter differences. The median value of the L4–L5 AP diameter difference in the hypoplasia group was significantly higher than in the control group (p<0.001), and the L5–S1 AP diameter difference was found to be significantly higher in the hypoplasia group than in the control group (p<0.001).

In the hypoplasia group, the wedging angle in the L5 vertebral body was found to be significantly higher than in the control group (p<0.001) (Table I). While there was no significant difference between the hypoplasia and control groups in terms of the anterior height of the L5 vertebra (p=0.768), the posterior height of this vertebra was significantly lower in the hypoplasia group (p<0.001) (Table II). The hypoplasia group was observed to have a significantly higher posterior epidural fat tissue thickness than the control group (p<0.001) (Table I).

Paired groups were compared according to the modified Pfirrmann grading system as low grade (grades 1-4) and high grade (grades 5-8) at the L4–L5 and L5–S1 intervertebral disc levels. It was observed that high grades were more common in the hypoplasia group (p<0.001) (Table III). According to the modified Pfirrmann scoring system, the percentage of grade 5-8, which indicates greater disc degeneration, was 50.9% in the group with vertebral body hypoplasia at the L4–L5 intervertebral disc level, and 16.3% in the control group (p <0.001) (Table III). In the group with vertebral body hypoplasia at the L5–S1 intervertebral disc level, the percentage of grade 5–8 was significantly higher (65.5% versus 32.7%; p<0.001) (Table III).

The rate of Modic signal changes in the L5 vertebra superior endplate was 34.5% in the hypoplasia group and 11.3% in the control group (p<0.001) (Table IV). The distribution of Modic signal changes in the inferior endplate of the L5 vertebra were higher in the hypoplasia group (p<0.001) (Table IV). The rate of spondylolysis was 7.7% in the control group and 65.5% in the hypoplasia group (p<0.001), and the rate of spondylolisthesis was 2.7% and 18.2%, respectively (p<0.001) (Table V).

## DISCUSSION

The condition in which the sagittal diameter of the vertebra is less than that of the upper vertebra is referred to as hypoplasia

**Table II:** Modified Pfirrmann Disc Degeneration Grading System

| Grade | Structural characteristics-signal intensity | Annulus Fibrosus-Nucleus Distinction | Disc height |
|---|---|---|---|
| **1** | Like cerebrospinal fluid, homogenously hyperintense | Present | Normal |
| **2** | Like presacral fat tissue, hyperintense | Present | Normal |
| **3** | Less hyperintense than presacral fat tissue | Present | Normal |
| **4** | Minimally hyperintense relative to the outer fibers of the annulus | Absent | Normal |
| **5** | Like the outer fibers of the annulus, hypointense | Absent | Normal |
| **6** | Hypointense | Absent | Less than 30% loss |
| **7** | Hypointense | Absent | 30-60 loss |
| **8** | Hypointense | Absent | More than 60% loss |

**Table III:** Comparison of Grades in Pfirrmann and Modified Pfirrmann Classification Systems in Hypoplasia and Control Groups

| | | Hypoplasia Group (n=55) | | Control Group (n=300) | | | |
|---|---|---|---|---|---|---|---|
| | | Number | % | Number | % | $c^2$ | *P |
| **L4-L5 disc level Pfirmann grading system** | **1** | 10 | 18.2 | 188 | 62.7 | 40.2 | **<0.001** |
| | **2** | 36 | 65.5 | 98 | 32.7 | | |
| | **3** | 6 | 10.9 | 11 | 3.7 | | |
| | **4** | 2 | 3.6 | 2 | 0.7 | | |
| | **5** | 1 | 1.8 | 1 | 0.3 | | |
| **L4-L5 disc level Modified Pfirmann grading system** | **1** | 0 | 0.0 | 18 | 6.0 | 45.8 | **<0.001** |
| | **2** | 5 | 9.1 | 91 | 30.3 | | |
| | **3** | 5 | 9.1 | 80 | 26.7 | | |
| | **4** | 17 | 30.9 | 62 | 20.7 | | |
| | **5** | 19 | 34.5 | 36 | 12.0 | | |
| | **6** | 6 | 10.9 | 10 | 3.3 | | |
| | **7** | 3 | 5.5 | 3 | 1.0 | | |
| **L5-S1 disc level Pfirmann grading system** | **1** | 10 | 18.2 | 155 | 51.7 | 26.9 | **<0.001** |
| | **2** | 28 | 50.9 | 88 | 29.3 | | |
| | **3** | 10 | 18.2 | 46 | 15.3 | | |
| | **4** | 4 | 7.3 | 4 | 1.3 | | |
| | **5** | 3 | 5.5 | 7 | 2.3 | | |
| **L5-S1 disc level Modified Pfirmann grading system** | **1** | 0 | 0.0 | 10 | 3.3 | 31.6 | **<0.001** |
| | **2** | 3 | 5.5 | 80 | 26.7 | | |
| | **3** | 8 | 14.5 | 67 | 22.3 | | |
| | **4** | 8 | 14.5 | 45 | 15.0 | | |
| | **5** | 19 | 34.5 | 44 | 14.7 | | |
| | **6** | 10 | 18.2 | 44 | 14.7 | | |
| | **7** | 4 | 7.3 | 5 | 1.7 | | |
| | **8** | 3 | 5.5 | 5 | 1.7 | | |

* *Chi-square test.*

**Table IV:** Distribution of Modic Signal Changes in L5 Vertebra Superior and Inferior Endplate from Hypoplasia and Control Group

| | | Hypoplasia Group (n=55) | | Control Group (n=300) | | $c^2$ | *P |
|---|---|---|---|---|---|---|---|
| | | Number | % | Number | % | | |
| Superior endplate | None | 36 | 65.5 | 266 | 88.7 | 25.2 | <0.001 |
| | 1 | 0 | 0.0 | 4 | 1.3 | | |
| | 2 | 19 | 34.5 | 29 | 9.7 | | |
| | 3 | 0 | 0.0 | 1 | 0.3 | | |
| Inferior endplate | None | 30 | 54.5 | 229 | 76.3 | 23.2 | <0.001 |
| | 1 | 2 | 3.6 | 6 | 2.0 | | |
| | 2 | 22 | 40.0 | 56 | 18.7 | | |
| | 3 | 1 | 1.8 | 9 | 3.0 | | |

**Chi Square test.*

**Table V:** Comparison of Spondylolysis and Spondylolisthesis Percentages Between the Study Groups

| | | Hypoplasia Group (n=55) | | Control Group (n=300) | | $c^2$ | *P |
|---|---|---|---|---|---|---|---|
| | | n | % | n | % | | |
| Spondylolysis | Absent | 19 | 34.5 | 277 | 92.3 | 112.0 | <0.001 |
| | Present | 36 | 65.5 | 23 | 7.7 | | |
| Spondylolisthesis | Absent | 45 | 81.8 | 292 | 97.3 | 23.2 | <0.001 |
| | Present | 10 | 18.2 | 8 | 2.7 | | |

**Chi-square test.*

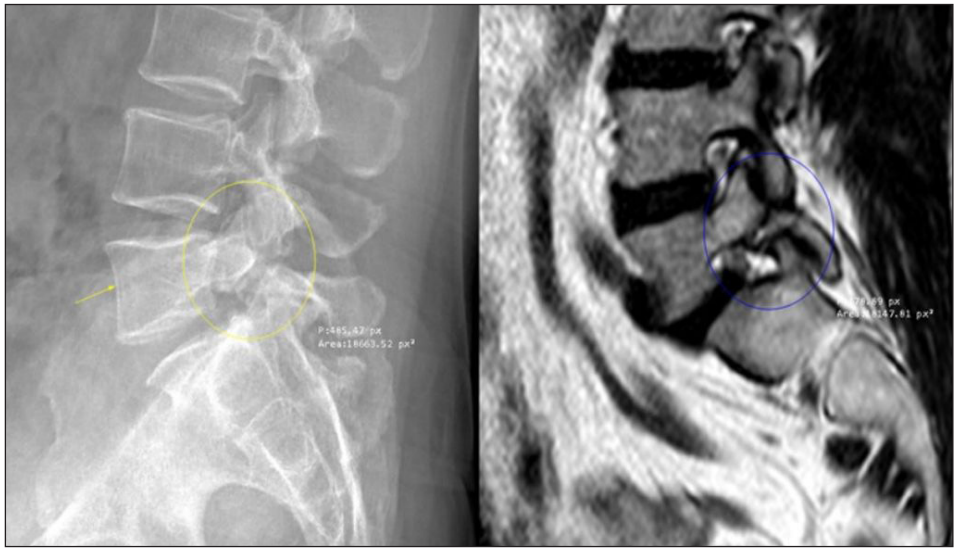

**Figure 3:** Lumbal lateral x-ray and T2-weighted sagittal plane MR images of a 42-year-old male patient; yellow and blue circle spondylolysis.

or short vertebra (21,23,30). Wilms et al. determined hypoplasia by measuring the A-P diameters of the L4 and L5 vertebrae in the midvertebral zone, and the A-P diameter of the S1 vertebra at the superior endplate level on midsagittal MRI images (31). Niggemann et al. detected hypoplasia by taking measurements at the level of the inferior endplate at L5 and the superior endplate of the S1 vertebra (21). Although the two studies adopted different methods for the determination of vertebral hypoplasia, the hypoplastic vertebrae rates were found to be similar. In the present study, the measurements

were made as described by Wilms et al. at the entrance of the basivertebral vein for the A-P diameters of L4 and L5, and at the midsagittal level of the superior endplate for the A-P diameter of the S1 vertebra. We found the frequency of vertebral body hypoplasia to be 1.8% in the 20-50 years aged population who presented with low back pain, which is similar to the rate reported by Wilms et al.

Different views have been reported in literature on the etiopathogenesis of vertebral body hypoplasia, with different studies interpreting it as hypoplastic dysplasia along with a congenital variant (32), while another suggests that it occurs due to such acquired reasons as radiation trauma (30). It has been stated that vertebral body hypoplasia develops over time as a result of repetitive trauma,while wedging causes spondylolysis in the posterior elements (10,19). Fractures occurring in the pars interarticular is that prevent the normal development of the vertebral body may cause hypoplasia (30). In a study by Wilms et al. involving cases with L5 vertebral body hypoplasia, a thinning of the inferior and posterior intervertebral joints of the peduncle was identified (30). Weakness in the posterior elements can lead to spondylolysis in this patient group. In our study, the rate of spondylolysis was 65.5% in patients with vertebral body hypoplasia, supporting the hypothesis that spondylolysis develops after hypoplasia.

In the vertebrae, wedging increases toward the caudal, and lumbar lordosis thus occurs, andit has been reported that wedging in the vertebral body increases more in cases with spondylolysis at the L5 level (2,21,30). Wiltse suggested that this excessive wedging may be an expression of hypoplastic dysplasia in the vertebral body (32). In the present study, the wedging angle in the L5 vertebral body was found to be significantly higher in the hypoplasia group than in the control group, and so the variation expressed as wedge vertebrae is considered to include also hypoplastic vertebrae. An increased wedging angle also leads to postural disorders in which the vertical tension vector, defined as a hyperlordotic vertebra, passes over the posterior elements (22). The wedging angle being significantly higher in patients with vertebral body hypoplasia in our study supports the view that hypoplasia poses a risk in terms of the degenerative changes that may occur in weak posterior elements and at the pars interarticularis level (20).

It has been shown that abnormal stress or loads in the vertebral endplate lead to histological changes in the adjacent bone marrow, and result in Modic signal changes (1). A Modic type 1 signal change reflects the inflammatory response that occurs with oedema and vascularization. In large cohort studies, it has been stated that type 1 Modic changes are associated with lower back pain (11), and that Modic type 2signal changes are associated with the formation of reactive bone tissue and granulation tissue (26). Modic type 3 signal change, on the other hand, is known to indicate sclerosis in the vertebral endplate (24). It has also been reported that the natural transformation of a type 1 signal change to a type 2 signal change takes two to three years (29). In the present study, evaluations were made based on the Modic signal change observed predominantly in the vertebral endplate. When the Modic signal changes seen in the superior and inferior endplates of the L5 vertebra were categorized, significant differences were noted between the hypoplasia and control groups ($p<0.001$). Kim et al. reported the radial pressure per unit area of the vertebral endplate to be higher in patients with vertebral body hypoplasia, and so it is not an unexpected finding that endplate degeneration occurs at a higher rate in patients with hypoplasia as the vertebral body area decreases (13). That said, the presence of more Modic signal changes at the inferior endplate of the L5 vertebra compared to the superior endplate in both the hypoplasia and control groups supports the view that degeneration is more prominent in the L5–S1 segment (12). We believe this situation may be a result of the higher mobility of the L5–S1 segment and the less stabilization in the posterior elements than at the other levels.

Lumbar spondylolysis is characterized by bone defects of the pars interarticularis, and occurs in approximately 6% of the adult population (16). In the present study, a similar rate of 7.7% was recorded in the symptomatic population with a mean age of 37.8 years. The relationship between bilateral spondylolysis and vertebral body hypoplasia at the L5 vertebra was first demonstrated in 1979 in a study based on conventional radiographs (7). Wilms et al. stated hypoplasia of the L5 vertebral body to be a common finding in patients with bilateral spondylolysis,and that there was no real shift in some of the cases evaluated as spondylolisthesis, using the term pseudospondylolisthesis to refer to this group (30). Niggemann et al. reported vertebral body hypoplasia being detected at a rate of approximately 42% in patients with isthmic spondylolisthesis (21). In the present study, among those with vertebral body hypoplasia, the frequency of spondylolysis was found to be 65.5%, and the frequency of spondylolisthesis was 18.5%. When compared to the normal population with lower back pain, a significant increase in the frequency of spondylolysis and spondylolisthesis was noted in patients with vertebral body hypoplasia.

Posterior intervertebral joint degeneration is a leading cause of back pain, especially in the elderly. Posterior intervertebral joint osteoarthritis classically includes both degenerative and proliferative features, such as a narrowing of the joint space, subarticular bone erosion, subchondral cysts, osteophyte formation and hypertrophy of the articular appendage. Early changes in degeneration begin in the articular cartilage, synovium and capsule, while in later stages the subchondral bone and joint space are affected (18). The capsule of each posterior intervertebral joint is richly innervated by the dorsal rami of the spinal nerve roots originating in adjacent levels (6). Changes due to degeneration lead to tension in the capsule and a narrowing of the joint space, resulting in pain. Butler et al. reported the prevalence of osteoarthritis in the posterior intervertebral joint to be 21% in the patient group with back pain, while Fujiwara et al. reported that osteoarthritis findings were observed at a rate of 38% in the case group (5,8,28). In the present study, grade 1–3 joint degeneration in the posterior intervertebral joints at the level of the L4–L5 vertebrae was found to be 32% on the right side and 28% on the left side in a symptomatic population with a mean age of 37 (20–50) years.

In the case group with vertebral body hypoplasia, which has been shown to be a predisposing factor for disc degeneration, this rate was found to be 74.5% on the right side and 70.1% on the left side. In the group with hypoplasia, degeneration in the posterior intervertebral joint was found to be significantly higher than in the control group, as would be expected.

MRI has limitations in detecting spondylolysis when compared to CT and radiography, although it has been suggested that secondary MRI findings, such as epidural fat interposition and reactive bone marrow signal changes, as well as increased imaging quality, can result in a higher sensitivity in the detection of spondylolysis using this imaging modality (27). In the present study, an evaluation was performed involving the correlation of the MRI, radiography and CT findings. It is difficult to detect early changes in posterior intervertebral joint osteoarthritis, such as minor chondral changes and synovial inflammation, on routine MRI, and this imaging method also has limitations in determining the severity of osteoarthritis due to the partial volume effect and the chemical shift artefact.

## CONCLUSION

Vertebral body hypoplasia is a predisposing factor for disc degeneration, posterior intervertebral joint osteoarthritis and endplate degeneration, and patients with vertebral body hypoplasia are more likely to develop spondylolysis and spondylolisthesis.

## AUTHORSHIP CONTRIBUTION

**Study conception and design:** HC, AT, HK

**Data collection:** HC, HK, VK

**Analysis and interpretation of results:** HC, AT, HK

**Draft manuscript preparation:** HC, HK

**Critical revision of the article:** AT, VK

All authors (HC, AT, HK, VK) reviewed the results and approved the final version of the manuscript.

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