# The Role of the Wedge Vertebra, Short Vertebral Body, Multifidus Muscle Atrophy in Isthmic Spondylolisthesis; an MRI Study

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#### ABSTRACT

**Objective:** We aimed to establish the frequency of short vertebral body, wedge vertebra and lumbar multifidus muscle atrophy in patients with isthmic spondylolisthesis and bilateral spondylolysis and to evaluate their places in the physiopathology.

*Material and Methods:* This study was planned retrospectively with 1400 patients who had a lumbar Magnetic Resonance Imaging; 44 patients with isthmic spondylolisthesis and 35 patients with bilateral spondylolysis but without spondylolisthesis and control group (n:44) were examined. Data analysis was performed using the NCSS 10 program. Statistical significance was set as p < 0,05.

**Results:** The percentage wedging was significantly larger in the 1. and 2. groups than in the control subjects (p<0.05). While we detected short vertebral body in 13 patients from 35 patients with spondylolysis, we established this in only one patient from the control group (n:44). The frequency of short vertebral body is statistically obviously lower in group 3 than the other two groups (p<0.001). LMM atrophy was seen in excess in the patients with isthmic spondylolisthesis when compare with the other groups (p<0.001).

**Conclusion:** Wedge vertebra, short vertebral body and multifidus muscle atrophy due to chronic degeneration and overload on hyperlordotic spine, are mostly seen together.

Keywords: isthmic spondylolisthesis, lumbosacral area, multifidus atrophy, short vertebral body, wedging vertebra

## INTRODUCTION

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Spondylolysis is defined as a defect in the pars interarticularis, whereas spondylolisthesis means slipping of a vertebra in relation to an adjacent vertebra. Spondylolisthesis can occur as a complication of spondylolysis due to a loss of posterior stabilisation in the affected segment <sup>(1,2)</sup>. The pathogenesis of spondylolysis and ÖΖ

İsthmik Spondilolisteziste Kama Vertebra, Kısa Vertebra ve Multifidus Kas Atrofisinin Rolü; Magnetic Rezonans Görüntüleme çalışması

**Amaç:** Amacımız isthmik spondilolistezis ve bilateral sspondilolizisli hastalarda kısa vertebra, kama vertebra ve lomber multifidus kas atrofi sıklığını saptamak ve fizyopatolojik süreçteki yerni değerlendirmek.

*Gereç ve Yöntemler:* Lomber Manyetik Rezonans Görüntüleme yapılmış 1400 hastada retrospektif olarak planlanmıştır; isthmik spondilolistezisli 44 hasta, spondilolistezisi bulunmayan 35 bilateral spondilolizisli hasta ve kontrol grubu (n=44) incelendi.Veri analizi NCSS 10 programında yapıldı. İstatistiksel anlamlılık için p<0.05 alındı.

**Bulgular:** Kamalaşma oranı 1 ve 2. grupta kontrol grubuna göre yüksekti (p<0.05). Kısa vertebra 35 spondilolizisli hastanın 13'te saptanırken kontrol grubunda (n=44) sadece 1 hastada saptandı. Kısa vertebra görülme sıklığı 3. grupta diğer iki gruba göre azdı (p<0.001). Multifidus kas atrofisi isthmik spondilolistezisli grupta diğer iki gruba göre belirgin olarak daha sık izlenmektedir (p<0.001).

**Sonuç:** Kronik dejenerasyon ve hiperlordotik omurgada aşırı yüklenmeye bağlı olarak kama vertebra, kısa vertebra ve multifidus kas atrofisi sıklıkla birikte izlenmektedir.

Anahtar kelimeler: isthmik spondilolistezis, kama vertebra, kısa vertebra, lumbosakral bölge, multifidus atrofisi

isthmic spondylolithesis is complex and the factors leading to spondylolysis and spondylolithesis are not fully understood. We aimed to establish the frequency of short vertebral body, wedge vertebra and lumbar multifidus muscle (LMM) atrophy in patients with istmic spondylolisthesis and bilateral spondylolysis and to evaluate their places in thephysiopathology.

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## **MATERIALS and METHODS**

This study began after the ethics committee approval.

#### Study population

1400 MRI scans of the lumbar spine acquired for various reasons between May 2014 and December 2014 at our facility were reviewed retrospectively. 35 patients with spondylolysis and 44 patients with isthmic spondylolisthesis were identified and included in the study. We selected that 44 patients with age- and sexmatched control group. For the control group only patients without spondylolysis or spondylolisthesis were included. All other patients were excluded from the study. Screening for inflammatory or malignant diseases such as metastasis or lymphoproliferative diseases were excluded, as well as posttraumatic indications because all of these diseases could affect the size of the lumbar vertebrae.

## MRI protocol

The MRI system was an 1,5 T MR unit (Signa HDxt; GE) and a body spine surface coil was used. Slices were positioned from T1 median sagittal images to the posterior caudal corner of the upper vertebral body and perpendicularly to the surface of the lumbar muscles. Only the lower level (L5-S1) was included in the analyses. Sagittal T1-W FSE, T2-W FSE and an axial T2-W FSE (3680/128 repitition time/echo time, 180x256 matrix, 280-mm field of view and 4-mm section thickness, NEX 2) was imaged for the study.

#### Image evaluation

All evaluations of lumbar MRI were evaluated in consensus by two radiologists (11 and 12 years of experience).

We examined the images specifically for the presence or absence of spondylolysis and spondylolisthesis at L5-S1. The measurement of spondylolisthesis is based on the widely recognized method proposed by Meyerding <sup>(3)</sup>. Meyerding defined the slippage on plain X-ray imaging in accordance to the vertebra below. The caudal vertebra is divided into four parts. Grade I means a translation of the cranial vertebra of up to 25%, Grade II of up to 50%, Grade III of up to 75%, and Grade IV up to 100%. Grade V was added later, describing the ptosis of the cranial vertebra <sup>(3)</sup>.



Figure 1a,c. Axial (a,b,c) T2-weighted MR images show that of lumbar multifidus muscles atrophy. These were rated as grade 0 (<10%) if normal condition (a); grade 1 for slight (10-50%) fat infiltration (b), and grade 2 for severe (>50%) fat infiltration (c).

The height of the vertebral body at L5 was measured anteriorly and posteriorly, and the percentage wedging was calculated for allhypoplastic or short L5 vertebrae with the simple formula: anterior height minus posterior height, divided by anterior height, as used by Frank and Miller <sup>(5)</sup>.

The grades of LMM atrophy were evaluated at L5-S1. Their lumbar MRIs were visually analyzed semi-quantitatively for signs of LMM muscle atrophy. Fat infiltration of the LMM was visually graded using the standard criteria: "normal" for estimates of 0-10% fat within the muscle, "slight" for 10-50% fat and "severe" for 50-100% fat (Fig 1). The grading system was adapted from previous studies using MRI <sup>(6,7)</sup>.

#### Statistical Analysis

Normality of data was evaluated by the Shapiro Wilk test and histogram. Results are expressed as mean±standard deviation (for normal data), median, minimum, maximum (for non-normal data) and frequencies and percentages (for nominal data).

The comparison of the ages between groups was evaluated by using one-way analysis of variance (ANO-VA). Since no difference was found, multiple comparison (post hoc) was not done. The wedging variable was compared between groups by Kruskal-Wallis one-way variance analysis. Post hoc comparison was performed by using Dunn's test. The comparison of nominal variables in the group (sex, LMM atrophy, short vertebral body) was done by chi square test. Data analysis was performed using the NCSS 10 program. Statistical significance was set as P<0,05 (two tailed).

#### RESULTS

Totally 123 patients with isthmic spondylolisthesis, spondylolysis and control subjects were included in this study.

Group 1: Spondylolisthesis was diagnosed in 44 patients; (38 Meyerding Grade 1 and 6 Meyerding Grade 2. 11 were male and 33 were female. The mean age was  $48.3 \pm 10.33$ .

Group 2: Bilateral spondylolysis and without spondylolisthesis at L5 was diagnosed in 35 patients; there were 16 male and 19 female with a mean age of 50.46±12.78 years.

Group 3: As a control group was selected 44 patients, 11 male and 33 female with a mean age of  $51.34\pm10.14$  years.

There is no statistically meaningful difference between all three groups in terms of sex (p=0.081) and age (p=0.416).



Figure 2a,b. Midsagittal T2-weighted fast spin-echo images show thatshort vertebral body at L5; reduced anteroposteriordiameter of L5 with posterior wedging. Grade1 anterolisthesis L5 on S1. The intervertebral disk at L5-S1 is dehydrated (a). Measurement of percentage wedging on the same image: Anterior height of L5 is 27.2 mm. Posterior height of L5 is 17.8 mm. Percentage wedging is 34% (b).



Figure 3a,b. Midsagittal T2-weighted fast spin-echo images; L5 vertebra which detected spondylolysis; percentage wedging is 27% (a) and difference in AP diameter is measured as 2.28 mm (b).

Difference in Anteroposterior Diameter:

Short vertebral body was detected in 10 patients of group 1 (n=44) (Figure 2A), in 13 patients of group 2 (n=35) and in 1 patient of group 3 (n=44). The frequency of short vertebral body is statistically obviously lower in group 3 than the other two groups (p<0.001).

## Percentage Wedging:

In the 1. group, the median percentage posterior wedging was 25.0% (range, 9-48%) (Figure 2b).



Figure 4. Midsagittal T2-weighted fast spin-echo image; L5 vertebra which spondylolisthesis was not detected; percentage wedging was 22%, two differences were shown in the measurement of midvertebral and lower vertebral margin.

Table 1. The distribution of short vertebral body, percentage wedging and lumbar multifidus muscle (LMM) atrophy for all three groups.

	Short Vertebral Body	Wedging	LMM Atrophy
1. Group (n.44)	10	25%	93,20%
2. Group (n.35)	13	24%	51,40%
3. Group (n.44)	1	20%	52,30%

In the 2. group, the median percentage posterior wedging was 24.0% (range, 17-46%) (Figure 3).

In the 3. group, the median percentage posterior wedging was 20.0% (range, 3-37%).

The percentage wedging was significantly larger in the 1. and 2. groups than in the control subjects (p<0.05). The percentage wedging was over 22% in 10 patients of the control group (n=44) and in 24 patients of group 2 with spondylolysis (n=35).

Fat infiltration of the LMM was detected 93.2% in the 1. group, 51.4% in the 2. group and 52.3% in the control group. LMM atrophy is more frequent significantly in group 1 than in group 2 and 3 (p<0.001) (Table).

## DISCUSSION

On the spine, the vertebral body height increases cau-

dally, wedging of lumbar vertebrae increases caudally too. These create the lumbar lordosis <sup>(8)</sup>. As a result, the lower lumbar vertebrae have a variant form of wedging, in addition to this to progress of the percentage wedging to the caudally on the spine <sup>(9)</sup>. Roussouly and Franco <sup>(10)</sup> described that in the hyperlordotic spine, the posterior arches of vertebral body are thinner, in this way, allows widely motion than before, especially in extension. Unfortunately this weakness of posterior arch is fragile. In the hyperlordotic spine, centre of gravity is on the posterior elements <sup>(10)</sup>.

In this study, it was established that in groups 1 and 2 the median percentage posterior wedging was similar and it was higher than in the control group (p<0.05). The ratio of posterior wedging increases with the appearance of spondylolysis.

After that the difference in length between the lower margin of L5 and upper margin of S1. A difference in length between the two vertebrae was recorded. Only differences equal to or greater than 3 mm was called vertebral hypoplasia. Niggemann et al.<sup>(4)</sup> was submitted that MRI allows the measurement of real distances without distortions and summation with overlying structures, and therefore real distances should be measured. When doing so, however, the image resolution must be taken into account. Because of the size of the field of view and the chosen resolution, a clear pointto-point discrimination is only possible at a distance of 3mm. Therefore, a slip or a hypoplasia of less than 3mm had to be discarded as it was not certain whether it was pathological or not. Therefore, we accepted 3 mm as cut off value for the hypoplasia diagnosis and Mayerding spondylolisthesis classification. However, since the etiopathogenesis is unclear, we preferred to use the term short vertebral body instead of the term hypoplasia.

Wilms G. et al. <sup>(11)</sup> submitted that 'L5 hypoplasia is a frequent finding in patients with bilateral spondylolysis.' Niggemann et al. <sup>(4)</sup> submitted that vertebral hypoplasia is a common diagnosis as 42% of their cases with isthmic spondylolisthesis. Although in both studies the detection rates of hypoplasic vertebra were very close to each other, it is seen that in these studies different methods were used to measure hypoplasia. Wilms et al. <sup>(11)</sup> calculated anteroposterior diameter L5 vertebrae was measured at the mid-vertebral zone on the midsagittal MR images. For the measurement of S1 vertebrae, they used upper vertebral surface on the same MR plan. On the other hand, Niggemann et al. (4) compared the measures of the inferior end plate of L5 and superior end plate of S1. Although for the normal vertebra corpus the measurements of mid-vertebral and lower vertebral margins are close to each others, it is obvious that the difference will increase especially for the wedge vertebra. By the increase in the percentage wedging the mid-vertebral diameter becomes shorter (Fig 4). In our study, since we think that the surfaces facing each other are more important we applied the end plate measurement which Niggemann et al. used. In our study short vertebral body was seen in one patient in the control group (group 3). The frequency of short vertebral body in the group 3 was obviously less than the group 1 and 2 (p<0.001). The congenital hypoplasia could be the first pathology. Correspondingly, it would cause thinning of the pedicles and elongation, which made them more vulnerable than the normal pedicles. The higher rate of short vertebral body in the groups 1 and 2 confirms also an opposite hypothesis; spondylolysis become initial. Short vertebral body develops frequently depending on spondylolysis. In the literature, studies related with spondylolysis defects established in adolescents are present. Morita et al. (12) said that spondylolysis is caused by repetitive micro trauma in the growing child. Ikata et al. (13) researched in adolescents with spondylolysis at lumbar region and they noticed that high percentage wedging of the L5 vertebra is not detected at early period however developed gradually in patients with spondylolysis. Different from our study, Ikata did not detect wedging at the early stages; this can be attributed to inclusion of exclusively adolescents in this study.

From paraspinal muscles, LMM providing stabilization at L5/S1 level was searched intensively. Kader et al. <sup>(7)</sup> detected in their study; LMM atrophy was seen in 80% of the cases with LBP and added the interrelationship between LMM atrophy and LBP. In our study, LMM atrophy is seen more frequently in the group 1 (spondylolisthesis group) when compared with the other two groups (p<0.001). When we take age and sex into account there is no statistically meaningful difference between all three groups (age p=0.416, sex p=0.081) and by eliminating the age and sex factor we can say LMM atrophy increase in connection with biomechanical problems and limited movements after slipping of the vertebra.

Our limitations; We think that using specific position (flexion-extension) scanning, weight bearing MRI instead of static MRI will be more reliable in the diagnosis and grading of spondylolisthesis. However, in our study we tried to overcome this limitation by correlating with the flexion-extension graphics of the patients who we established spondylolysis.

## CONCLUSION

The posterior wedge vertebra is a main developmental pathology on hyperlordotic spine and it can be seen without trauma and spondylolysis. Spondylolysis could occurr on the hyperlordotic spine as a result of the increased load over the posterior elements due to wedging. Since the frequency of short vertebral body was statistically significantly higher in the patients with spondylolysis and spondylolisthesis, relation with the end plate degeneration is possible. It was seen that LMM atrophy was added to the process later.

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