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ORIGINAL STUDY

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The effect of transitional vertebrae and spina bifida occulta on disc herniation, disc degeneration, and end-plate changes in pediatric patients with low back pain

Ozge Gulsum Illeez, Fatma Esra Bahadir Ulger, Ilknur Aktas

From the Fatih Sultan Mehmet Training and Research Hospital, Istanbul, Turkey

The aim of the study is to investigate the assumption whether lumbosacral transitional vertebrae (LSTV) and spina bifida occulta (SBO) cause lumbar disc herniation (LDH), intervertebral disc degeneration (IDD), and vertebral endplate changes / Modic changes (MCs) in children and adolescents with low back pain (LBP). Four hundred patients (aged 10-17) with LBP persisting for at least six weeks were included in the study. Lumbosacral X-rays were examined for the presence of LSTV and SBO. The prevalence of IDD/MCs and LDH at L4-5 and L5-S1 levels were investigated by evaluating the lumbosacral MRI of the patients with and without LSTV-SBO. The study population consisted of 219 girls and 181 boys with mean age 14.9±1.9. LSTV was determined in 67 (16.8%) patients and SBO in 62 (15.5%). No significant difference was observed in the prevalence of IDD, MCs, and LDH in patients with and without LSTV/SBO. LSTV and SBO were not observed in approximately 80% of patients without LDH and IDD/MCs. The presence of LSTV and SBO does not appear to represent a risk factor for early degeneration in lumbar spine and LDH in children and adolescents with LBP.

Keywords: Transitional vertebrae; spina bifida occulta; disc herniation; disc degeneration; endplate changes; pediatric.

INTRODUCTION

Low back pain (LBP) is a widespread global health problem, not only in adults but also in the pediatric age group. Epidemiological studies indicate that the prevalence of LBP in adolescents is very consistent with that seen in adults (18-51%) *(1)*.

Lumbosacral transitional vertebra (LSTV) and spina bifida occulta (SBO) are the most frequently encountered congenital structural malformations in lumbar region. Although their association with LBP is controversial, one study reported that these malformations may be an underlying cause of pediatric nonspecific LBPs (mechanical LBP) *(2)*. LSTV is defined as a complete or partial unilateral or bilateral fusion to the sacrum of the enlarged transverse process of the lowest lumbar vertebra. LSTV has been implicated in hypomobility at this level and in hypermobility at the upper level.

■ Ozge Gulsum Illeez, MD,
■ Fatma Esra Bahadir Ulger, MD,
■ Ilknur Aktas, Professor, Dr
University of Health Sciences, Fatih Sultan Mehmet Training and Research Hospital, Istanbul, Turkey.
Correspondence: Ozge Gulsum Illeez, E5 Karayolu Üzeri. 34752. İçerenköy-Ataşehir. İstanbul/Türkiye. Phone: +9053262 55684, Fax: +902165750406.
Email : ozgeilleez@hotmail.com

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Clinical studies in adults have demonstrated that hypermobility can result in disc degeneration and facet joint changes and in a higher degree of herniation at the upper level of the transitional vertebra (L4-5) than at other levels *(3-5)*. SBO is the result of unsuccessful fusion between the posterior vertebral elements with no impact on the spinal cord or meninges. This defect is generally observed in the L5 and S1 vertebrae. SBO has been implicated as leading to LBP due to instability of the base of the lumbar spine**.**

As in adulthood, LBP in the pediatric age group may have various causes. Although it is seen less frequently than adults, one of these causes is lumbar disc herniation (LDH). Adult LDH is believed to result from repetitive overloading and age-related degenerative changes. Age-related degeneration results in a loss of load-bearing ability in the lumbar intervertebral joint, the disc, the ligaments, and the facet joints. Failure in the supporting structures leads to rupture of the annulus fibrosus under stress, in turn giving rise to disc herniation and related symptoms *(6)*. Spinal degenerative changes are believed to not occur in children and adolescents under normal conditions. Although serious degeneration is actually rare, the onset of degenerative changes in the intervertebral disc occurs before the second decade of life in pediatric cases *(2)*. The risk factors for LDH and early spinal degeneration in childhood are still not fully elucidated because these two entities are less common than adulthood.

Could LSTV and SBO be the cause of LDH, IDD and MCs in pediatric age group with low back pain? Our study aims to find an answer to this question.

MATERIALS AND METHODS

The medical records of all patients aged 10-17 admitted to our department from June 2013 to October 2019 with LBP were reviewed retrospectively. Four hundred eighty-two patients with LBP persisting for six weeks or more were identified from 1267 patients presenting with LBP. Patients with malignancy, spondylolysis-listhesis, spinal alignment disorders such as scoliosis and kyphosis, with inflammatory diseases and found to have sacroiliitis or high levels of acute phase reactants (ESR and CRP), or with histories of spinal surgery were excluded from the study, and 400 patients were finally enrolled. LSTV and SBO were evaluated using lumbosacral x-ray, while IDD, MCs and LDH were evaluated using magnetic resonance imaging (MRI) with 1.5 Tesla, all assessments being performed by a specialist radiologist. While all patients had x-ray imaging, only 163 patients had MRI. LSTV was classified in line with Castellvi's radiographic system *(3)*. Type I A: unilateral dysplastic transverse process >19 mm in height. Type I B: bilateral dysplastic transverse process >19 mm in height. Type II A: enlarged transverse process with unilateral pseudoarthrosis with the adjacent sacral ala. Type II B: enlarged transverse process with bilateral pseudoarthrosis with the adjacent sacral ala. Type III A: unilateral lumbarization/sacralization with complete osseous fusion of the transverse processes to the sacrum. Type III B: bilateral lumbarization/sacralization with complete osseous fusion of the transverse processes to the sacrum. Type IV: Type II on one side and type III on the other (mixed type). The Modic classification was performed using T1 and T2 weighted sagittal MRI *(7,8)*. MCs are classified under three types. Type 1: MCs show decreased signal intensity on T1-weighted images and increased signal intensity on T2-weighted images. Type 2: MCs demonstrate increased signal intensity on both T1- and T2-weighted images, while Type 3: MCs reflect decreased signal intensity on both T1- and T2-weighted images. The Pfirrmann grading was performed using T2-weighted sagittal MRI *(9)*. Grade I: The disc is homogeneous with bright hyperintense white signal intensity and normal disc height. Grade II: The disc is inhomogeneous, although the hyperintense white signal is preserved. The nucleus and annulus are clearly differentiated, and a horizontal gray band may be observed. The disk is normal in height. Grade III: The disc is inhomogeneous with an intermittent gray signal intensity. The distinction between the nucleus and annulus is unclear. The disc is normal or slightly less than normal in height. Grade IV: The disc is in homogeneous with a hypointense dark gray signal intensity. No more distinction remains between the nucleus and annulus. The disc height is slightly

or moderately decreased. Grade V: The disc is inhomogeneous with a hypointense black signal intensity. No more distinction remains between the nucleus and annulus. The disc space is collapsed. Pfirrmann grades I-II were regarded as normal and grades III-IV-V were regarded as degenerated disc. Local ethical committee approval was obtained for this study.

RESULTS

The study population consisted of 400 patients aged between 10 and 17 years (mean 14.9±1.9, median 15). The number of girls (n=219, 54.8%) was slightly higher than that of boys (n=181, 45.2%). SBO was determined in 62 (15.5%) patients, and LSTV in 67 (16.8%) (figure 1). According to Castellvi classification; Type 1a in 6 patients with 1.5% of cases, type 1b in 3 patients with 0.8%, type 2a in 15 patients with 3.8%, type 2b in 24 patients with 6.0%, type 3b in 13 patients with 3.3%, type 4 in 6 patients with 1.5%. The most common type was LSTV type 2 (n=39, 9.8%).

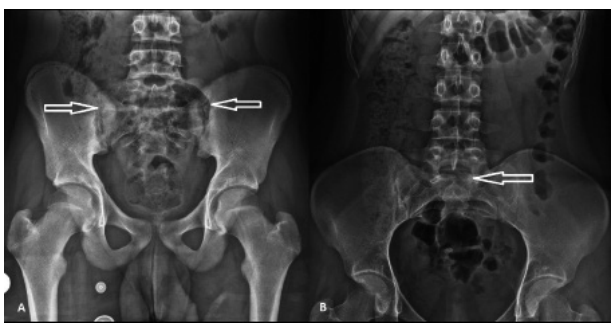

Figure 1. — (A) Radiography of one patient with type 2b LSTV and (B) radiography of another patient with SBO.

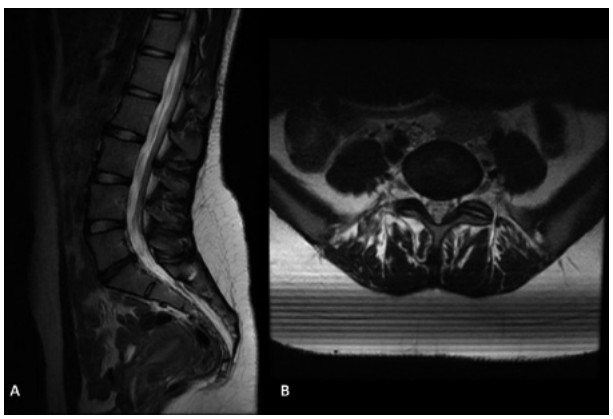

Figure 2. — (A) sagittal t2w, (B) axial t2w images showing lumbar (spinal) MRI of the patient with central protrude herniation at L5-S1 level.

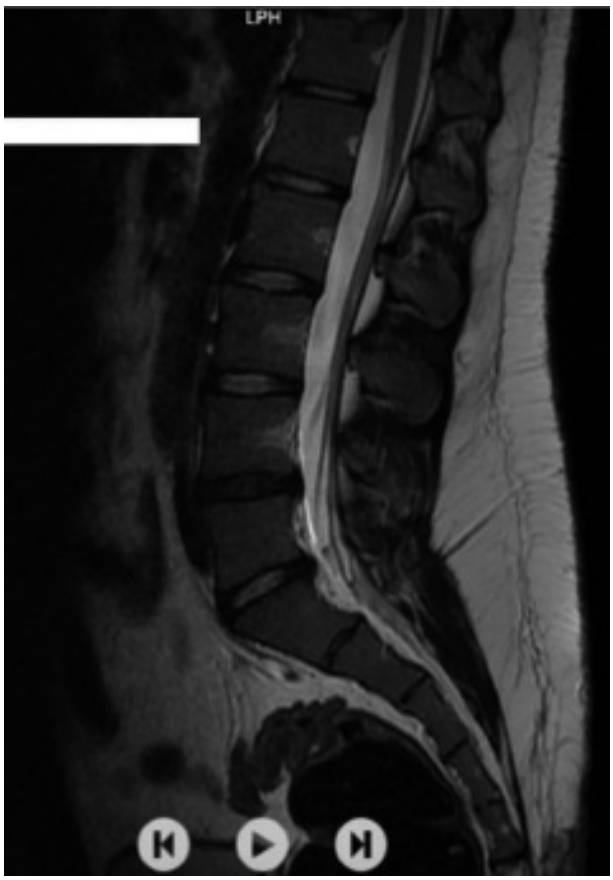

Figure 3. — Sagittal t2w image of the patient with type 4 intervertebral disc according to Pfirrmann grading system at L4-5 level.

LDH (protrusion+extrusion) was detected in 68 of the 163 patients undergoing MRI (L4-5 n=18, 26.5%, L5-S1 n=33, 48.5%, and simultaneously at both levels n=17, 25%) (figure 2), MCs at the L4-5 level in 14 (8.6%), MCs at the L5-S1 level in 39 (23.9%), IDD at the L4-5 level in 21 (12.8%) (figure 3), and IDD at the L5-S1 level in 31 (19%) (table I). No significant difference was observed between patients with and without LSTV in terms of prevalences of LDH, IDD and MCs at L4-5 and L5-S1 ($p>0.05$) (table II). No significant difference was also observed between patients with and without

Table I. — Distribution of type of LDH, level of LDH, MCs at L4-5 and L5-S1 and IDD at L4-5 and L5-S1 parameters in patients undergoing magnetic resonance imaging

		n	%
Type of LDH(n=163)	**None**	12	7.4
	Bulging	83	50.9
	Protrusion	66	40.5
	Extrusion	2	1.2
Level of LDH(n=68)	**L4-5**	18	26.5
	L5-S1	33	45.8
	L4-5, L5-S1	17	25.0
MCs L4-L5 (n=163)	**None**	149	91.4
	Type 1	1	0.6
	Type 2	13	8.0
MCs L5-S1 (n=163)	**None**	124	76.1
	Type 1	1	0.6
	Type 2	32	19.6
	Type 3	6	3.7
IDD L4-L5 (n=163)	**Grade1**	118	72.4
	Grade2	24	14.7
	Grade3	9	5.5
	Grade4	10	6.1
	Grade5	2	1.2
IDD L5-S1 (n=163)	**Grade1**	102	62.6
	Grade2	30	18.4
	Grade3	15	9.2
	Grade4	9	5.5
	Grade5	7	4.3

SBO in terms of prevalences of LDH, IDD and MCs at L4-5 and L5-S1 ($p>0.05$) (table III). Results concerning the diagnostic value of LSTV and SBO in LDH and IDD/MCs are given in table IV. Regardless of age and gender, IDD was observed to significantly increase LDH risk (table V).

Statistical analysis was performed on IBM SPSS v24 software. Descriptive statistics were expressed as mean ± standard deviation and median (min-max). Qualitative data were expressed as percentages. Comparisons between qualitative data were performed using Pearson's Chi-square and Fisher's exact tests. Sensitivity, specificity, and negative and positive predictive values were calculated for LSTV/SBO and combinations thereof in order to analyze the relation between these anomalies and LDH and IDD/MCs. These values were expressed at a 95% confidence interval, and confidence intervals were determined using Wilson's method and continuity correction. p values <0.05 were regarded as statistically significant. The determinants of LDH was assessed with binary logistic regression. The adjusted variables are age, gender, existence of MCs and IDD at levels L4-L5 and L5-S1. The significancy of the model was tested with omnibus test of coefficient and Hosmer and Lemeshow test.

DISCUSSION

Consistent with the general literature, the prevalence of LSTV in the present study was 16.8%, and that of SBO was 15.5%. In agreement with Castellvi et al., the most common LSTV type in this study was type 2. Type 2, together with type 4, is also thought to be associated with LBP *(10)*. No significant difference was detected in the prevalences of LDH and IDD/MCs in patients with and without LSTV/SBO. Investigation of the diagnostic value of LSTV and SBO revealed higher specificity and negative predictive values than sensitivity and positive predictive values. Eighty percent or more of patients without LDH and IDD/MCs were found not to have LSTV and SBO. In addition, approximately 80% of patients without these malformations also did not have these pathologies. This finding suggests that paying more attention may not required in terms of LDH and IDD/MCs in children and adolescent patients without LSTV and SBO.

The relation between LSTV/SBO and LBP, IDD, and LDH in the adult age group is controversial, and no consensus has been achieved *(5,11-13)*. There have been few studies about LSTV and SBO in the pediatric age group, and those that have been performed have largely consisted of few patients receiving surgical treatment. One study detected congenital anomalies including lumbarization, sacralization, and SBO in 20 out of 70 children and adolescents operated for disc herniation *(14)*. Another study implicated early onset IDD, congenital

Table II. — Presence of LDH and MCs L4-L5 and L5-S1 and IDD L4-5 and L5-S1 parameters depending on the presence of LSTV

		Presence of LSTV		
		No	Yes	
		n (%)	n (%)	Test value, p
Presence of LDH	**No**	80 (57.6)	15 (62.5)	0.206, 0.650*
	Yes	59 (42.4)	9 (37.5)	
MCs L4-L5	**No**	127 (91.4)	22 (91.7)	1.000**
	Yes	12 (8.6)	2 (8.3)	
MCs L5-S1	**No**	107 (77.0)	17 (70.8)	0.425, 0.515*
	Yes	32 (23.0)	7 (29.2)	
IDD L4-L5	**No**	121 (87.1)	21 (87.5)	1.000**
	Yes	18 (12.9)	3 (12.5)	
IDD L5-S1	**No**	113 (81.3)	19 (79.2)	0.782**
	Yes	26 (18.7)	5 (20.8)	

*Pearson Chi-square, **Fisher's exact test.

Table III. — Presence of LDH and MCs L4-L5 and L5-S1 and IDD L4-5 and L5-S1 parameters depending on the presence of SBO

		Presence of SBO		Test value, p
		No	Yes	
		n (%)	n (%)	
Presence of LDH	**No**	78 (57.8)	17 (60.7)	0.082, 0.774*
	Yes	57 (42.2)	11 (39.3)	
MCs L4-L5	**No**	121 (89.6)	28 (100.0)	0.131**
	Yes	14 (10.4)	0 (0.0)	
MCs L5-S1	**No**	101 (74.8)	23 (82.1)	0.684, 0.408*
	Yes	34 (25.2)	5 (17.9)	
IDD L4-L5	**No**	117 (86.7)	25 (89.3)	1.000**
	Yes	18 (13.3)	3 (10.7)	
IDD L5-S1	**No**	108 (80.0)	24 (85.7)	0.492, 0.483*
	Yes	27 (20.0)	4 (14.3)	

*Pearson Chi-square, **Fisher's exact test.

lumbosacral anomalies, and recurring trauma in the etiopathogenesis of 101 patients operated due to LDH *(15)*. Skeletal scintigraphy was performed on 48 patients in one study investigating the relation between LSTV and LBP in the pediatric population, and high uptake at the transverse-sacral articulation was observed in approximately 80%. It is also reported that stress at the transverse-sacral articulation could contribute to LBP *(16)*. In their study of 63 patients aged 10-19 operated for disc herniation, Dang et al. observed at least one congenital anomaly in the lumbosacral region in 60 patients, and reported higher levels of herniation at the L4-5 level in patients with sacralization and

Table IV. — The diagnostic value of LSTV and SBO in LDH and IDD/MCs in adolescents with LBP

	Disc herniation			
	Sensitivity % (95% C.I.)	**Specificity % (95% C.I.)**	**Positive predictive value % (95% C.I.)**	**Negative predictive value % (95% C.I.)**
LSVT (+)	13.2 (6.6-24.1)	84.2 (74.9-90.6)	37.5 (19.5-59.2)	57.6 (48.8-65.8)
SBO (+)	16.2 (8.7-27.5)	82.1 (72.6-88.9)	39.3 (22.1-59.3)	57.8 (48.9-66.1)
LSVT + SBO (+)	0.0 (0.0-6.7)	69.5 (59.1-78.3)	0.0 (0.0-6.9)	57.9 (48.3-66.9)
	Vertebral degeneration L4-L5			
	Sensitivity % (95% C.I.)	**Specificity % (95% C.I.)**	**Positive predictive value % (95% C.I.)**	**Negative predictive value % (95% C.I.)**
LSVT (+)	14.3 (2.5-43.8)	**85.2 (78.3-90.3)**	8.3 (1.5-28.5)	**91.4 (85.1-95.3)**
SBO (+)	0.0 (0-26.8)	**81.2 (73.8-86.9)**	0.0 (0.0-15.0)	**89.6 (82.9-94.0)**
LSVT + SBO (+)	0.0 (0.0-26.8)	68.5 (60.3-75.7)	0.0 (0.0-6.9)	89.5 (82.0-94.2)
	Vertebral degeneration L5-S1			
	Sensitivity % (95% C.I.)	**Specificity % (95% C.I.)**	**Positive predictive value % (95% C.I.)**	**Negative predictive value % (95% C.I.)**
LSVT (+)	17.9(8.1-34.1)	**86.3 (78.7-91.6)**	29.2 (13.4-51.2)	**77.0 (68.9-83.5)**
SBO (+)	12.8 (4.8-28.2)	**81.5 (73.3-87.7)**	17.9 (6.8-37.6)	**74.8 (66.5-81.7)**
LSVT + SBO (+)	0.0 (0.0-11.2)	70.2 (61.2-77.9)	0.0 (0.0-6.9)	76.3 (67.3-83.6)
	Disc degeneration L4-L5			
	Sensitivity % (95% C.I.)	**Specificity % (95% C.I.)**	**Positive predictive value % (95% C.I.)**	**Negative predictive value % (95% C.I.)**
LSVT (+)	14.3 (3.8-37.4)	**85.2 (78.0-90.4)**	12.5 (3.3-33.5)	**87.1 (80.0-91.9)**
SBO (+)	14.3 (3.8-37.4)	**82.4 (74.9-88.1)**	10.7 (2.8-29.4)	**86.7 (79.5-91.7)**
LSVT + SBO (+)	4.8 (0.3-25.9)	69.0 (60.6-76.3)	33.3 (1.8-87.5)	86.0 (77.9-91.5)
	Disc degeneration L5-S1			
	Sensitivity % (95% C.I.)	**Specificity % (95% C.I.)**	**Positive predictive value % (95% C.I.)**	**Negative predictive value % (95% C.I.)**
LSVT (+)	16.1 (6.1-34.5)	**85.6 (78.2-90.9)**	20.8 (7.9-42.7)	**81.3 (73.6-87.2)**
SBO (+)	12.9 (4.2-30.8)	**81.8 (73.9-87.8)**	14.3 (4.7-33.6)	**80.0 (72.1-86.2)**
LSVT + SBO (+)	0.0 (0.0-13.7)	69.7 (61.0-77.2)	0.0 (0.0-6.9)	80.7 (72.0-87.3)

higher levels of herniation at the L5-S1 level in patients with lumbarization *(6)*. Similarly, Zhang et al. compared patients aged 14-20 operated for LDH with healthy peers and reported a significant relation between LSTV and LDH, and that LDH at the L4-5 level was significantly higher in patients with sacralization *(17)*.

LDH is regarded as a pathology that develops during IDD *(18)*. Consistent with that thesis, LDH was more common in patients with IDD in the present study. The biodynamics and anatomy of the lumbar spine alter over time. When all spinal structures are gradually degenerated, the connective tissues of the intervertebral discs, the nucleus pulposus, and the annulus fibrosus lose their elasticity, and annular tears and fissures occur in the last plaques with changes in collagen structure. These changes can lead to disc herniation *(18)*. Due to age-related changes, adults are more at risk in terms of disc herniation than children and adolescents. The estimated incidence is between 0.1% and 0.2% of the entire pediatric population *(19)*. Disc herniation

Table V. — Determinants of LDH with logistic regression analysis

Variables	Odds Ratio	95% C.I.	P value
Age			
10-14	**Ref**		
15-17	**1.0**	**0.5-2.2**	**0.995**
Gender			
Girl	**Ref**		
Boy	**1.2**	**0.6-2.6**	**0.581**
MCs L4-L5			
No	**Ref**		
Yes	**0.1**	**0.02-1.1**	**0.060**
MCs L5-S1			
No	**Ref**		
Yes	**1.6**	**0.5-5.1**	**0.431**
IDD L4-L5			
No	**Ref**		
Yes	**9.8**	**1.9-50.3**	**0.007**
IDD L5-S1			
No	**Ref**		
Yes	**11.1**	**2.9-42.5**	**<0.001**

Omnibus test of coefficient chi-square=44.5, p<.001, Hosmer-Lemeshow test chi-square=5.1, p=0.533, Nagelkerke r2=0.322.

in this age group has been more associated with trauma and/or active sporting activities *(20)*. Other implicated factors include congenital malformations such as LSTV and SBO, genetic factors, epiphyseal ring separation and spinal sagittal alignment *(21)*. MRI was performed on patients with persistent LBP in the present study, and LDH was detected in 42%. This high rate may be due to our investigating a selected population, to the frequent engagement in sporting activities among the patients in our study group, and to traumatic cases not being excluded.

Studies in the literature have reported frequent delays in the diagnosis of disc herniation in the pediatric age group, this being attributed to much less neurological deficit being initially observed, despite the clinical findings not differing markedly from those in adults *(22)*. The possibility of disc herniation in children and adolescents may not be considered as much as in adults or it may be overlooked in patients without having a neurological deficit. Detailed evaluation and advanced radiological investigations are therefore recommended for definite diagnosis in the pediatric age group *(22)*.

In terms of early degeneration, although no significant relation was observed in the present study, due to their frequent occurrence, malformations such as LSTV and SBO, genetic factors, systemic diseases, overweight/obesite, enviromental determinants and hormonal influences have been implicated in early degeneration *(23-25)*.

Onset of IDD probably occurs in childhood. One cadaver study reported a prevalence of IDD of 6-16% between the ages of 10 and 19 years *(26)*. Another study observed IDD at one or more levels in 33% of 15-year-old participants with and without LBP. That study also described IDD as representing an increased risk for recurrent LBP both following the rapid growth period and in young adulthood *(27)*. In their study of patients aged 14-15 years, Tertti et al. described IDD as a common finding in both patients with LBP and in the control group, and reported that degeneration was most commonly associated with disc protrusion *(28)*. Another study reported IDD in 61 out of 69 patients with non-specific LBP in the 8-16 age group, LSTV (38 sacralisation and 17 lumbarisation) in 55, and SBO in 31 *(2)*. Based on previous studies, MCs are known to be common in the middle and advanced age, but it is rare in the pediatric age group and in young adults. *(24)* In the present study, however, Modic Type 2 changes were detected in L5-S1 at a level of approximately 20%.

The present study has some limitations. Some of the patients' information could not be reached due to its retrospective nature and only patients with low back pain were investigated without having comparison with the healthy control group. The reason for our lack of healthy control group is that we do not want to expose participants in this age group to radiation. So we established the study design only on patients. Also, since all of our patients did not have MRI, LSTV-SBO, LDH and early degeneration relationship could be studied in a limited number of patients. The reason for the low number of advanced imaging is that the most common type of LBP in this age group is non-specific self-limiting LBP. Therefore, patients' complaints were taken under control during follow-up and there was no need for MRI.

CONCLUSION

The present study suggests that LSTV and SBO are not risk factors for LDH and early degeneration in lumbar spine in children-adolescent with low back pain. In the pediatric age, comprehensive, prospective studies should be planned to investigate the effects of LSTV / SBO on lumbar biomechanics and other risk factors that may cause LDH / early spinal degeneration.

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