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Original Investigation

Demographical Aspects of Central Large Lumbar Disc Herniation

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ABSTRACT

AIM: The purpose of this study was to investigate the risk factors for the development of the central large disc herniations and to compare the demographic data between central mass prolapse and broad-based central disc herniation.

MATERIAL and METHODS: Between 2002 and 2007, 1630 patients underwent surgery and a large disc herniation was the main problem in 59 patients (3.6%). We performed a retrospective analysis of the demographic data of these patients. Magnetic resonance (MR) images were evaluated according to the disc type and level. Variables were evaluated both at baseline and follow-up, with special emphasis on physical job characteristics, sports activities, and MR - based morphologic findings.

RESULTS: Central large disc herniation was diagnosed in 59 patients consisting of 41 males and 18 females. The average age was 34.7 years. 36 patients had a central mass prolapse that occupied more than 50% of the spinal canal. Intraoperative observations confirmed that 29 out of 36 central disc prolapse patients (80.5%) had intact posterior longitudinal ligaments. Interestingly, the condition in these 29 patients was found to have a direct relation with age and occupation or other body training sports activities.

CONCLUSION: The size of the large central disc herniation, physical activity, age and gender are major factors in the development of disc herniation.

KEYWORDS: Large lumbar disc herniation, Outcome, Physical Activity

INTRODUCTION

The incidence of central lumbar disc herniation (CLDH) is low as compared with the more common occurrence of paramedian or posterolateral disc herniations (6). However, there have been no studies that compare the demographic analyses of central mass prolapse (CMP) and contained central disc herniations (CCDH).

The etiology of degenerative spine disease is multifactorial and includes; cumulative effects of microtrauma to the spine, osteoporosis, smoking, excess body weight and loss of muscle tone. Especially excess body weight effects stresses on the spine. On the other hand, loss of abdominal and paraspinal muscle tone results in increased dependence on the bony spine for structural support (19). In accordance with the literature, there is no definitive information regarding the incidence or prevalence of disc herniations in a sizable population that would define the norm (5, 45). Driving, sedentary occupation, vibration, smoking, previous full-term pregnancy, physical inactivity, increase in body mass index (BMI), and tall stature can be some of the risk factors for disc herniation (37). BMI increase and physical activity are also reported to be associated with higher incidence of lumbar disc herniation in the young (38).The increased number of adults and adolescents who regularly participate in athletic activities has raised the collective awareness of common low-back injuries such as bruising, over stretching, or mild to moderate tearing of the paraspinal soft tissues (13). Prevalence varies among sports participation and further varies depending on the position played (12, 35). Taken together, the annulus,

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disc, and posterior elements bear significant combinations of tensile stress and compressive and shear force, respectively, whereas the posterior soft tissues bear considerable resistive stress. These forces are coincided in varying degrees in all athletic activities (15, 16). Intuitively, one might assume that physical fitness would be preventive with regard to disc rupture.

The term, "degenerative spine disease" may be preferable to "degenerative disc disease". This is a progressive deterioration of the structures of the spine. Lumbar disc herniation involve both biomechanical and biochemical changes (40). Disc degeneration begins with breakdown of aggrecan molecule which are present in both the nucleus pulposus and annulus fibrosus in the mature disc. The degenerating disc contains high proportions of non-aggregating proteoglycans which are unable to interact with hyaluronan molecules. This leads to decreased water content within the nucleus. Loss of hydration of the nucleus pulposus results in stress transmission to the periphery rather than the centre of the end-plates, resulting in increased size of the end-plates and decreased range of motion. A defect in the annulus fibrosus can result in disc herniation (40). No matter which definition is used, disc "degeneration" is mostly in the lower lumbar spine and increases with age (47). Genetic inheritance has the highest risk with an approximately 50-70% of variability in disc degeneration between identical twins (7, 8, 41). Environmental influences are probably reflected by it and on spinal level this rate does not include the strong intrasubject dependence of disc degeneration (3, 8). High and repetitive mechanical loading are the environmental risk factors for disc herniation (3, 9). Heavy lifting is claimed to be closely associated with disc prolapse, but not with other features of spinal degeneration or age which suggest that the prolapse is not an integral part of the aging process (9, 43).

Our study was conducted to compare the demographic features of the patients and evaluate the association between occupational exposure and morphological magnetic resonance imaging (MRI) findings of CLDH.

■ MATERIAL and METHODS

During the period from November 2002 through December 2007, 1630 patients were operated on for lumbar disc herniation in our department of neurosurgery. We retrospectively researched archives of these patients, and we detected 59 patients (3.6%) among the 1630 who were operated on for symptomatic CLDH that were radiographically verified. Occupations and habituations of the patients were evaluated separately. The mean follow-up period was 15 months (range 12 to 24 months).

We made use of the evaluation charts which all the patients in our department had been asked to fill out regarding personel, medical, and occupational histories. Data of the follow-up visits were reviewed to evaluate the long-term outcome. In addition, neurological conditions and demographic data of the patients so as to gender, age, job, history of the diseases and intraoperative observation notes were obtained.

In the present study, the criteria for definition of CLDH were based on MRI. All patients had preoperative noncontrast MRI of the lumbar spine. A 1.5-Tesla magnetic resonance (MR) scanner (Vision; and AvantoSiemens, Erlangen, Germany) was used for all patients. The MR sequences included axial and sagittal T1 and T2 images from which the measurements were obtained. The images were 4 mm thick with a 10 mm interslice gap. The matrix differs from 240x320 to 307x384 in sagittal T2 W, 200x384 to 320x320 in sagittal T1 W images, 200x384 to 230x384 in axial T2 W images and 166x320 to 166x320 in axial T1 W images. The field of view was 30cm for sagittal images and 25 cm for the axial images All MR images were analyzed independently by a neuroradiologist who documented the disc type and level of involvement.

Using MRI, we classified the patients with CLDH as CMP and CCDH as described by Bärlocher et al. (6):

1) CMP with extrusion or sequestration of disc material, in general, occupying more than 50% of the sagittal midline diameter (Figure1A-C)
2) CCDH with an intact PLL and occupying less than 50% of the sagittal intraspinal diameter (Figure 2A-C). The apex of a CLDH should be located in the midline of the spinal canal with a tolerance of 10% for deviation. Sequestrated unilateral disc fragments were not enrolled in this study.

MRI is generally considered to be the most sensitive noninvasive method for examining disc degeneration and biochemical changes in the disc at present (34). Degenerative changes were evaluated according to Modic's classification of MR imaging characteristics (19). T1 and T2-weighted signals have been demonstrated to correlate with biochemical changes in the nucleus pulposus associated with aging and disc degeneration. In the original Modic's classification, loss of T1-weighted signal intensity (hypointensity) and marked T2-weighted signal intensity (hyperintensity) are described as a degenerative disc change of Modic Type 1. Marked T1 and T2-weighted signal intensity (hyperintensity) are described as a degenerative disc change of Modic Type 2. Loss of T1 and T2-weighted signal intensity (hypointensity) are described as a degenerative disc change of Modic Type 3, because it suggests general biochemical changes in the disc. Evaluation of the integrity of the PLL was based on the MR findings described by Grenier et al. (20).

Frequencies, rates, arithmetic means, standard deviations were used in descriptive statistical analysis. Chi square test was applied for discrete data, and t test was applied for the indiscrete data. Spearman's rho coefficient was used to evaluate relations. Results were evaluated in the confidence interval of 95% and significance was considered as $p<0.05$. Data were analyzed using SPSS –PC version 15.0 (statistical Program for Social Science, v. 15.0 for Windows, SPSS, Evanston, IL).

■ RESULTS

The study population consisted of 59 patients (41 men, 18 women), who had undergone an operation with the diagnosis of CLDH with a mean age of 34.96 years (range 21-72 years). 36 patients had CMP with extruded (17 patients) or sequestered (19 patients) disc material and 23 patients had CCDH. Patients with CMP comprised of 7 females and 29 males with mean age of 29.19 years (range, 21-47).Patients with CCDH comprised of 11 females and 12 males with mean age of 44 years (range, 31-72). Mean BMI was 23.7 in CMP and 29.2in CCDH patients. Difference between CMP and CCDH regarding age and BMI was found statistically significant. The characteristics of the 59 patients are shown in Table I. Demographic data and occupational facilities are reported in Table II.

Occupations of the 41 male patients included sedentary work (clerical), hard labor (that requires physical strength), active military service (commando, etc), sportsman (9 patients), and regular sports at leisure time. Most of the female patients were housewives. 4 patients had sedentary occupations, one patient was hard laborer and 3 patients worked at physical education centers.

The symptomatic disc levels were L3/4 in 7 patients, L4/5 in 31 patients and L5/S1 in 21 patients. Comparison between levels did not reach any statistical significance.

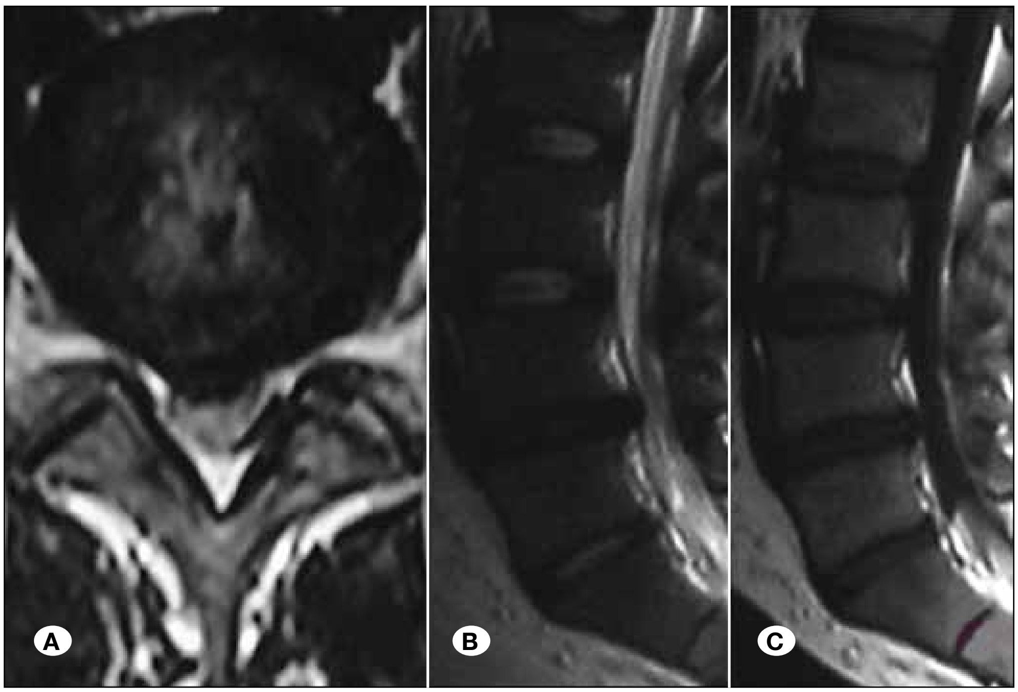

Figure 1: A) T2-weighted axial MR image that reveals central mass prolapse occupying more than 50% of the canal.
B, C) T2 and T1 weighted sagittal MR images of the same patient with mass prolapse in the central of the canal. Disc material is not extruded.

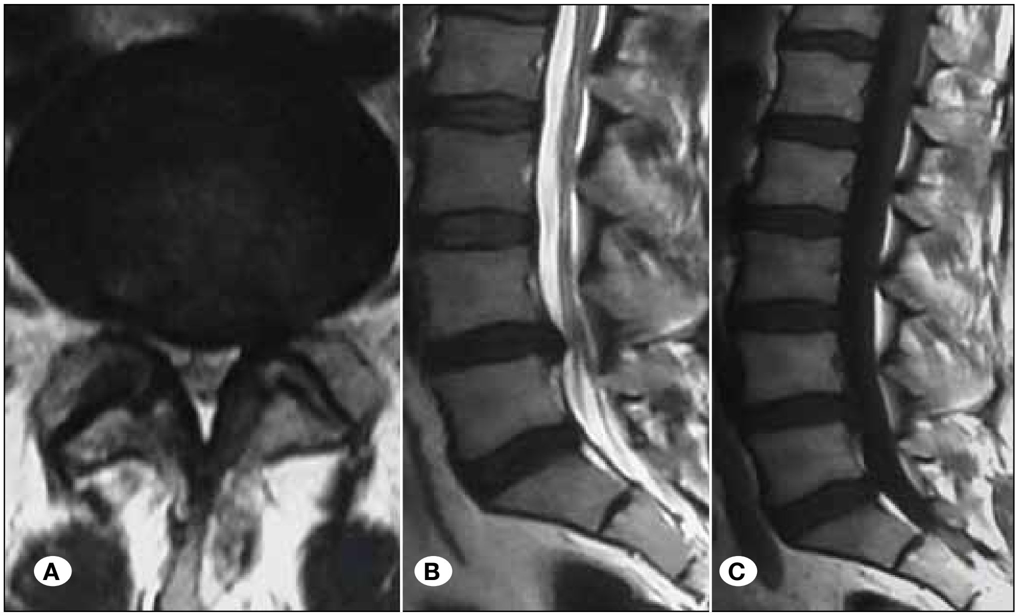

Figure 2: T2 Weighted **(A)** axial MR image, **(B, C)** T2- and T1-weighted sagittal MR images that reveals contained central disc herniations with an intact PLL and occupying less than 50% of the sagittal intraspinal diameter.

Table I: Predictor Variables Obtained from Questionnaires at Baseline in Central Lumbar Disc Herniation (n=59)

Variable	Patients	
Sociodemographic data		
Age	mean 34.7 (21-72) years	
Gender		
Male	41 (69.4%)	
Female	18 (30.5%)	
Anthropometric data		
	CMP	**CCDH**
Weight (kg)	mean 73.7	85.1
Height (centimeter)	mean 176	170.5
Body mass index (kg/m²)	mean 23.7 (19.2-29)	29.2 (24.6-34)
MRI findings		
Level of Disc herniation		
L3-4	7 (11.9%)	
L4-5	31 (52.5%)	
L5-S1	21 (35.6%)	
Type of the disc herniation		
Extruded (CMP)	17 (28.8%)	
Sequestered (CMP)	19 (32.2%)	
Contained (CCDH)	23 (39%)	
Work schedule		
Sedentary work	11 (18.6%)	
Hard labor	9 (15.2%)	
Army officer	11 (18.6%)	
Sportman	9 (15.2%)	
Regular sports	6 (10.1%)	
Housewife	10 (16.9)%)	
Gymnast	3 (5.1%)	

Table II: Comparing Overall Patient Demographic and Occupational Data Between CMP and CCDH

Type of disc herniation	Total	BMI	Mean age (year)	Female / Male	Female-Occupation	Male- Occupation
CMP	36	23.7 (19.2-29)	29.19 (21-47)	7 (19.4%) / 29 (80.5%)	3 (16.6%) Gymnast 4 (22.2%) Sedentary work	11 (26.8%) Elite military job 9 (21.9%) Sportman 6 (14.6%) Regular sports 3 (8.3%) Sedentary work
CCDH	23	29.2 (24.6-34)	44 (31-72)	11 (47.8%) / 12 (52.1%)	10 (55.5%) Housewife 1 (5.5%) Hard labor	4 (17.3%) Sedentary work 8 (19.5%) Hard labor
Total	59	25.9	34.96	18 (30.5%)/41 (69.4%)	18	41

The process of CMP formation was positively associated with young age and BMI. CMP was also directly influenced by the sports practice (p=0.0001; r=0.79) (Table III). Housewives and sedentary workers were significantly overrepresented in CCDH (Table IV). As for the gender, CCDH and CMP were more frequent in women and men (Table III, IV). Male gender was positively and BMI was negatively related to type of disc herniation (CMP or CCDH) (p=0.001; r=0.85 / p=0.016; r=0.56). Correlation of the Spearman's rho coefficients of the occupational and level of CLDH variables yielded no significant association between them.

The degree of disc degeneration was significantly related to the type of degenerated discs and the extent of disc herniation at baseline (Table III, IV). Degenerative changes were found only in 10.1% of all patients and degenerations defined by Modic were detected only in 6 patients (26.08 %) with CCDH.

Having excluded the effects of age and BMI, it was found that hard labor and sedentary work had a statistically significant correlation with CLDH. Aging and BMI were directly associated with CCDH and inversely with CMP (p=0.012; r=0.33).

Operation records of all the 29 CMP patients confirmed that PLLs were intact, and according to their demographic data, they were all physically active patients. CCDH, the other subtype of CLDH, was significantly associated with less physical activity during leisure time. Presence of sports activities and being active military personnel (commando etc.) were significant independent predictors of progressive extension of disc herniation (Table III, IV).

■ DISCUSSION

The aim of present study was to analyze the relationship between occupational exposure and degenerative changes in the lumbar spine. As a secondary goal, we examined the effect of work-related risk factors on CLDH. Several risk factors related to physical aspects of the workplace such as heavy physical work, lifting and forceful movements, awkward posture and static work posture were suggested by results of previous studies. In previous studies, physical occupational risk factors have been reported to be responsible for the development of CLDH (10, 21, 29-31). We detected that the subtype of CLDH was closely related to the chronicity of physical activity and it was influenced directly by how heavy the workload was. This is a preliminary study to compare herniation location and morphology outcomes with demographic factors in surgically treated CCDH and CMP patients. There were various reports, which had shown strong predisposition to lumbar disc herniation due to trauma (14, 25, 26, 42, 44), while others showed little association (11, 25, 27). Later series mentioned that degenerative changes already existed and trauma only aggravated the disc herniation. In our series, 5% of patients had a history of significant trauma. Degenerative changes were seen only in 10.1% of patients.

18.6% of the patients who had lumbar disc herniation had a positive family history. This association was stated as 60-68 % (38, 44). Weak connective tissue or early predisposition of degenerative changes in the spine can play a role in

Table III: Characteristics of the Patients with CMP

Gender	Age	BMI	Level	Occupation
M	21	23	L4-5	AO
M	29	23	L5-S1	AO
M	25	23	L4-5	AO
M	27	23	L5-S1	AO
M	22	24	L4-5	AO
M	24	23	L4-5	AO
M	28	24	L5-S1	AO
M	28	25	L3-L4	AO
M	31	24	L4-5	AO
M	28	24	L5-S1	AO
M	21	24	L4-5	AO
M	26	25	L4-5	RS
M	35	24	L5-S1	RS
M	27	24	L4-5	RS
M	21	23	L4-5	RS
M	28	25	L4-5	RS
M	28	24	L5-S1	RS
M	23	22	L4-5	S
M	31	24	L3-L4	S
M	33	23	L5-S1	S
M	24	22	L4-5	S
M	27	23	L4-5	S
M	27	23	L3-L4	S
M	24	24	L4-5	S
M	26	24	L4-5	S
M	25	26	L4-5	S
M	37	25	L5-S1	SW
M	39	26	L5-S1	SW
M	42	25	L5-S1	SW
F	43	23	L5-S1	SW
F	35	26	L5-S1	SW
F	45	29	L4-5	SW
F	48	27	L5-S1	SW
F	23	21	L4-5	G
F	24	19	L4-5	G
F	27	19	L3-L4	G

***SW:** Sedentary work, **HL:** Hard labor, **AO:** Army officer, **S:** Sportman, **RS:** Regular sports, **H:** Housewife, **G:** Gymnast.*

Table IV: Characteristics of the Patients with CCDH

Gender	Age	BMI	Level	Occupation
F	42	24.6	L4-5	HW
F	45	27.9	L5-S1	HW
F	38	27	L3-4	HW
F	36	27.7	L4-5	HW
F	33	33.2	L4-5	HW
F	72	29	L5-S1	HW
F	49	31.1	L5-S1	HW
F	63	30.1	L5-S1	HW
F	65	29.7	L5-S1	HW
F	57	30.4	L4-5	HW
F	45	34	L5-S1	HL
M	38	29.1	L4-5	HL
M	38	30.1	L3-4	HL
M	33	27.8	L4-5	HL
M	31	28.7	L4-5	HL
M	37	29.1	L4-5	HL
M	40	28.7	L4-5	HL
M	37	29.4	L3-4	HL
M	46	29.7	L4-5	HL
M	48	29.4	L5-S1	SW
M	38	27.5	L4-5	SW
M	33	30.6	L4-5	SW
M	63	28.4	L5-S1	SW

***SW:** Sedentary work, **HL:** Hard labor, **AO:** Army officer, **S:** Sportman, **RS:** Regular sports, **H:** Housewife, **G:** Gymnast.*

these families. To exclude other damage, most studies have focused exclusively on lumbar disc degeneration (30). Any concurrent age-dependent lumbar spine degeneration can complicate the occupational determinants of these changes in an unbiased analysis. As a matter of fact, there are reports on direct and significant influences of aging on lumbar disc degeneration and end-plate defects (46). In the present study, several additional demographical determinants besides age were associated with morphological changes in the disc.

High BMI and a few sports activities are also reported to be associated with a higher incidence of lumbar disc herniation in adolescents (11, 38). On the other hand, in adults, disc herniation and end-plate defects show little correlation with age and they most commonly affect only the lower lumbar levels (47). In our series, L4-5 intervertebral disc space was the most commonly afflicted level. Regular physical activity appears to reduce or increase the risks of disc degeneration, depending on how severe it is (1). Sudden and large increases in mechanical loading may cause intervertebral discs to be particularly vulnerable to injury and fatigue damage because muscles and bones will be strengthened by increased physical loading faster than the avascular discs (2). Although some manual occupations carry no greater risk than sedentary work, these ideas could explain why frequent lifting of heavy loads causes the highest known risk factors for disc prolapse, either at work or in the home (22, 36). "Adaptive remodeling" in response to controlled exercise explains why physically active individuals have extremely strong vertebrae and discs (17, 18, 39). Conversely, lack of exercise leads to tissue weakening, and this may explain why some sedentary occupations increase the risk of disc prolapsed (22). The weakened spinal tissues would be vulnerable to accidental injury during slips and falls by which PLL encounters overloading that leads to CLDH. This strengthens the importance of the morphological changes that PLL might have undergone simultaneously with the soft tissues and the bones due to regular physical activity.

The PLL strongly attaches to the annulus fibrosis, and frequently is torn in cases of free fragment disc herniations (32). As the PLL (posterior longitudinal ligament) is not attached to the posterior surface of the vertebrae, the superficial and deep layers can be distinguished easily. A midline bony septum on the posterior surface of the vertebral body between the superior and inferior margins is the attachment site for both superficial and deep layers. Deficiencies in the septum in the central third of the vertebral body cause this attachment to not always be continuous. The central part of the superficial layer fibers extend to and merge with the anterior aspect of the dura mater (28). Herniated disc material would also tend to collect beneath the deep layer.

It has been stated by Putz et al. that all movements are associated with fibers from both layers, as the higher number of fibers are recruited with the higher load (28). A protective role is supported by the neighborhood of the PLL within the epidural space. The existence of an additional membranous structure (the superficial membrane of the PLL) associated with the PLL has been seen, although its morphology has been argued. It is obvious that a greater insight of the morphology and morphometry of the PLL and associated membranes throughout the length of the spine is necessary for determining its role in spinal biomechanics and protection (28). However, spine flexibility and linked range of motion have been proven to be reduced in degeneration due to the increasing size of the endplate and the decreasing height of the intervertebral disc space (28).

Decreased disc height of the nucleus with resultant loss of tension of the surrounding ligaments was found to cause increased laxity of the motion segment. (32). The role in dynamic motion of the spine is due the presence of elastin in the ligamentum flavum. A lot of elastic fibers are present in the normal dural layer and significantly fewer elastic fibers are present in the degenerated dorsal layer. Furthermore, with age, elastic fibers decrease on the dorsal side, but not on the dural side. Because of this, loss of elastic fibers mainly on the dorsal side results in a decrease in the elasticity of ligamentum flavum. Previously, it was biomechanically reported that mechanical stress is higher on the dorsal side compared with the dural side during lumbar motion (24). On the contrary, the loss of elastic fibers mainly at the dorsal side in PLL may be explained by the high mechanical load. Elasticity loss of PLL may result in folding into the neural canal and thus it could easily cause disconnection from the midline bony septum on the posterior surface of the vertebral body. Thus, to elucidate the pathomechanism of the sequestered disc herniation in the elderly, sedentary occupation, and physical inactivity, should be investigated. Consequently, the pathomechanism can be attributed to the qualitative changes in ligamentum flavum on the basis of loss of elasticity and strength. It is not difficult to assume then that the content of elastic fibers of PLL decreases with aging. However, there has been no study explaining in depth the pathomechanism of loss of elasticity. However, similar histologic changes were reported to occur at the insertion site of the medial collateral ligament of the knee joint, Achilles tendon and supraspinatus tendon (24). Thus, the qualitative changes of PLL may not occur with activity are due to mechanical loading, which would not induce degenerative changes resulting in decrease of PLL elasticity. Likewise, since the PLL of the physically active people like sportsmen and military personnel may actually have more elastic structure than the physically inactive ones, these people may have more tendency to develop central mass prolapse with an intact PLL. It means that elasticity of the PLL in these subjects causes the disc fragment to herniate to the center of the canal forming a large 'saccus' that occupies larger space in the central canal rather than tearing the PLL.

All the aforementioned probable changes in the PLL are assumed to be evaluated by MRI studies. The sensitivity, specificity, and accuracy of detecting the disruption of the PLL by MR images were reported as 71-100%, 78-92%, and 80-81%, respectively, compared with operative findings (4, 20, 23, 33). Not only MR images of the PLL were interpreted, but also the integrity of the PLL in the operative field was actually checked.

We observed that, CCDH was rare in the young and physically active people and though they had CLDH that occupied more than 50% of the canal, particularly, their PLL were intact in most of them by which they differed from the classical CMP definitions. According to our results, loss of elasticity of the PLL may play an important role in sequestered and large central disc herniation. Considering the fact that PLL plays key role in the development of CMP, it is therefore not surprising that this disorder is more common in athletic people.

■ CONCLUSION

The data obtained from the patient records, radiological evaluations and demographic analyses indicate that physically active people are more likely to develop CMP compared to CCDH without any ligamentous tear, owing to the more elastic PLL structure that they have. Though this information challenges the general opinion accepted so far, it is worth considering and needs to be confirmed in larger series of patients.

■ REFERENCES

1. Adams MA, Freeman BJ, Morrison HP, Nelson IW, Dolan P: Mechanical initiation of intervertebral disc degeneration. Spine 25(13):1625-1636,2000
2. Adams MA, Dolan P: Could sudden increases in physical activity cause degeneration of intervertebral discs? Lancet 350(9079):734-735, 1997
3. Adams MA, Roughley PJ: What is intervertebral disc degeneration, and what causes it? Spine 31(18):2151-2161, 2006
4. Ahn SH, Ahn MW, Byun WM: Effect of the transligamentous extension of lumbar disc herniations on their regression and the clinical outcome of sciatica. Spine 25(4):475-480, 2000
5. Baldwin NG: Lumbar disc disease: The natural history. Neurosurg Focus 13(2):E2, 2002
6. Barlocher CB, Krauss JK, Seiler RW: Central lumbar disc herniation. Acta Neurochir 142(12):1369-1374, 2000
7. Battié MC, Videman T, Gill K, Moneta GB, Nyman R, Kaprio J, Koskenvuo M: 1991 Volvo Award in clinical sciences. Smoking and lumbar intervertebral disc degeneration: An MRI study of identical twins. Spine 16(9):1015-1021, 1991
8. Battie MC, Videman T, Gibbons LE, Fisher LD, Manninen H, Gill K: Determinants of lumbar disc degeneration. A study relating lifetime exposures and magnetic resonance imaging findings in identical twins. 1995 Volvo Award in clinical sciences. Spine 20(24):2601-2612, 1995
9. Battie MC, Videman T, Parent E: Lumbar disc degeneration: Epidemiology and genetic influences. Spine 29:2679-2690, 2004
10. Bernard BP: Musculoskeletal disorders and workplace factors. A Critical Review of Epidemiologic Evidence for Work-Related Musculoskeletal Disorders of the Neck, Upper Extremity, and Low Back. Cincinnati: US Department of Health and Human Services, 1997
11. Boos N, Weissbach S, Rohrbach H, Weiler C, Spratt KF, Nerlich AG: Classification of age-related changes in lumbar intervertebral discs: 2002 Volvo Award in basic science. Spine 27(23):2631-2644, 2002
12. DePalma MJ, Bhargava A: Nonspondylolytic etiologies of lumbar pain in the young athlete. Curr Sports Med Rep 5(1):44-49, 2006
13. Dunn IF, Proctor MR, Day AL: Lumbar spine injuries in athletes. Neurosurg Focus 21(4):E4, 2006
14. Ebersold MJ, Quast LM, Bianco AJ: Results of lumbar discectomy in the pediatric patient. J Neurosurg 67(5):643-647, 1987
15. Farfan HF: Muscular mechanism of the lumbar spine and the position of power and efficiency. Orthop Clin North Am 6(1):135-144,1975
16. Farfan HF: The biomechanical advantage of lordosis and hip extension for upright activity. Man as compared with other anthropoids. Spine 3(4):336-342,1978
17. Granhed H, Jonson R, Hansson T: The loads on the lumbar spine during extreme weight lifting. Spine 12(2):146-149, 1987
18. Granhed H, Jonson R, Hansson T: Mineral content and strength of lumbar vertebrae. A cadaver study. Acta Orthopaedica Scandinavica 60(1):105-109, 1989
19. Greenberg MS: Handbook of neurosurgery. Low back pain and radiculopathy. 7th ed. New York, 2010:430
20. Grenier N, Greselle JF, Vital JM, Kien P, Baulny D, Broussin J, Senegas J, Caille JM: Normal and disrupted lumbar longitudinal ligaments: Correlative MR and anatomic study. Radiology 171(1):197 -205, 1989

21. Hoogendoorn WE, Bongers PM, de Vet HC, Douwes M, Koes BW, Miedema MC, Ariëns GA, Bouter LM: Flexion and rotation of the trunk and lifting at work are risk factors for low back pain: Results of a prospective cohort study. Spine 25(23):3087-3092, 2000
22. Kelsey JL, Githens PB, White AA 3rd, Holford TR, Walter SD, O'Connor T, Ostfeld AM, Weil U, Southwick WO, Calogero JA: An epidemiologic study of lifting and twisting on the job and risk for acute prolapsed lumbar intervertebral disc. J Orthop Res 2(1):61-66, 1984
23. Kim KY, Kim YT, Lee CS, Kang JS, Kim YJ: Magnetic resonance imaging in the evaluation of the lumbar herniated intervertebral disc. Int Orthop 17(4):241-244, 1993
24. Kosaka H, Sairyo K, Biyani A, Leaman D, Yeasting R, Higashino K, Sakai T, Katoh S, Sano T, Goel VK, Yasui N: Pathomechanism of loss of elasticity and hypertrophy of lumbar ligamentum flavum in elderly patients with lumbar spinal canal stenosis. Spine 32(25):2805-2811, 2007
25. Kumar R, Kumar V, Das NK, Behari S, Mahapatra AK: Adolescent lumbar disc disease: Findings and outcome. Childs Nerv Syst 23(11):1295-1299, 2007
26. Kurihara A, Kataoka O: Lumbar disc herniation in children and adolescents. A review of 70 operated cases and their minimum 5-year follow-up studies. Spine 5(5):443-451, 1980
27. Lee JY, Ernestus RI, Schroder R, Klug N: Histological study of lumbar intervertebral disc herniation in adolescents. Acta Neurochir 142(10):1107-1110, 2000
28. Loughenbury PR, Wadhwani S, Soames RW: The posterior longitudinal ligament and peridural (epidural) membrane. Clin Anat 19(6):487-492, 2006
29. Luoma K, Riihimaki H, Luukkonen R, Raininko R, Viikari-Juntura E, Lamminen A: Low back pain in relation to lumbar disc degeneration. Spine 25(4):487-492, 2000
30. Mariconda M, Galasso O, Imbimbo L, Lotti G, Milano C: Relationship between alterations of the lumbar spine, visualized with magnetic resonance imaging, and occupational variables. Eur Spine J 16(2):255-266, 2007
31. Marras WS, Lavender SA, Leurgans SE, Fathallah FA, Ferguson SA, Allread WG, Rajulu SL: Biomechanical risk factors for occupationally related low back disorders. Ergonomics 38(2):377-410, 1995
32. Martin MD, Boxell CM, Malone DG: Pathophysiology of lumbar disc degeneration: A review of the literature. Neurosurg Focus 13(2):E1, 2002
33. Masaryk TJ, Ross JS, Modic MT, Boumphrey F, Bohlman H, Wilber G: High-resolution MR imaging of sequestered lumbar intervertebral disks. AJR Am J Roentgenol 150(5):1155-1162, 1988
34. Möller A, Maly P, Besjakov J, Hasserius R, Ohlin A, Karlsson MK: A vertebral fracture in childhood is not a risk factor for disc degeneration but for Schmorl's nodes. Spine 32(22):2487-2492, 2007
35. Mundt DJ, Kelsey JL, Golden AL, Panjabi MM, Pastides H, Berg AT, Sklar J, Hosea T: An epidemiologic study of sports and weight lifting as possible risk factors for herniated lumbar and cervical discs. The Northeast Collaborative Group on low back pain. Am J Sports Med 21(6):854-860, 1993
36. Mundt DJ, Kelsey JL, Golden AL, Panjabi MM, Pastides H, Berg AT, Sklar J, Hosea T: An epidemiologic study of non-occupational lifting as a risk factor for herniated lumbar intervertebral disc. Spine (Phila Pa 1976) 18(5):595-602, 1993
37. Nachemson AL: Prevention of chronic back pain. The orthopaedic challenge for the 80's. Bull Hosp Jt Dis Orthop Inst 44(1):1-15, 1984
38. Pietila TA, Stendel R, Kombos T, Ramsbacher J, Schulte T, Brock M: Lumbar disc herniation in patients up to 25 years of age. Neurol Med-Chir 41(7):340-344, 2001
39. Porter RW, Adams MA, Hutton WC: Physical activity and the strength of the lumbar spine. Spine 14(2):201-203, 1989
40. Samandouras G: The Neurosurgeon's Handbook. Lumbar disc disease. New York: Oxford, 2010:830
41. Sambrook PN, MacGregor AJ, Spector TD: Genetic influences on cervical and lumbar disc degeneration: A magnetic resonance imaging study in twins. Arthritis Rheum 42:366-372, 1999
42. Schindler OS, Fairbank JC: Two-level intervertebral disc herniation in an adolescent. Br J Clin Pract 50(3):171-173, 1996
43. Seki S, Kawaguchi Y, Chiba K, Mikami Y, Kizawa H, Oya T, Mio F, Mori M, Miyamoto Y, Masuda I, Tsunoda T, Kamata M, Kubo T, Toyama Y, Kimura T, Nakamura Y, Ikegawa S: A functional SNP in CILP, encoding cartilage intermediate layer protein, is associated with susceptibility to lumbar disc disease. Nature Genetics 37(6):607-612, 2005
44. Shillito J Jr: Pediatric lumbar disc surgery: 20 patients under 15 years of age. Surg Neurol 46(1):14-18, 1996
45. Weber H: The natural history of disc herniation and the influence of intervention. Spine 19(19):2234-2238, 1994
46. Videman T, Nurminen M, Troup JD: Lumbar spinal pathology in cadaveric material in relation to history of back pain, occupation, and physical loading. 1990 Volvo Award in clinical sciences. Spine 15(8):728-740, 1990
47. Videman T, Battie MC, Gill K, Manninen H, Gibbons LE, Fisher LD: Magnetic resonance imaging findings and their relationships in the thoracic and lumbar spine. Insights into the etiopathogenesis of spinal degeneration. Spine 20(8):928-935, 1995