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ORIGINAL ARTICLE

Cigarette Smoking Dose as a Predictor of Need for Surgical Intervention in Patients with Lumbar Disk Herniation

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Purpose: Numerous studies have investigated the significant relationship between sciatic pain, radiating lower back pain, lumbosacral radicular syndrome or other disk disorders and cigarette smoking; however, only few reports have demonstrated the relationship between the total smoking dose and lumbar disk herniation (LDH), a cause of lower back or sciatic pain. Furthermore, the relationship between total cigarette consumption and the need of surgical intervention for LDH has not yet been investigated. **Materials and Methods:** This study included 391 patients with symptomatic LDH. The control group comprised 431 inpatients admitted for other medical or surgical problems. Their demographic data and level of cigarette consumption were obtained through a chart review. The association between lumbar surgical intervention and the clinical characteristics were investigated by multiple logistic regression analyses, with stepwise selection. **Results:** Compared with the nonsmokers, the smokers had a 1.5-fold increased risk of developing LDH ($P = 0.01$). An increased total smoking dose (pack-years) was a risk factor of undergoing lumbar surgical intervention among the LDH patients (odds ratio [OR] = 1.02; $P = 0.03$). Furthermore, the risk of undergoing lumbar surgical intervention increased to 1.83 times among LDH patients with a 6-10-year smoking history and to 2.16 times among those with >10-year smoking history ($P = 0.02$ and $P = 0.002$, respectively). **Conclusion:** This study found that the total cigarette smoking dose was associated with LDH and was a risk factor for undergoing surgical intervention for LDH.

Key words: herniated disk, cigarette smoking, smoking dose, surgical intervention, predictor

INTRODUCTION

Lumbar disk herniation (LDH) is a serious public health issue worldwide. It is a common problem resulting in lower back pain. Although not life-threatening, chronic lower back pain affects the patients' health status, limits the daily activities, and decreases the quality of life. Many reports show that such patients have a poor functional status and low quality of life.[1] Smoking is also an important issue because it is associated with many diseases, including, atherosclerosis, chronic infection,[2] cardiovascular disease, and cancer.[3-5] Many reports have shown a clear association between smoking and spinal diseases. Scott *et al*.,[6] Goldberg *et al*.,[7] and Hadley and Reddy[8] have reported that cigarette smoking is associated with lower back pain. Livshits *et al*.,[9] and Porter and Hanley[10] have proved that the smoking habit is significantly associated with degenerative disk disease. Among the cases of low back pain (LBP) and sciatica, 4-5% were caused by LDH.[11] However, previous studies have revealed an inconsistent correlation between smoking and LDH, and few reports have demonstrated a relationship between the total smoking dose and LDH, a cause of lower back or sciatic pain. Furthermore, none of these previous studies have investigated the relationship between the total smoking dose and the need of surgical intervention for LDH. Therefore, the objective of the present study is to investigate the relationship between the total smoking dose and LDH. In addition, after adjusting for the potential confounding

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variables, this study explores whether lumbar surgery for LDH is associated with the total smoking dose. It is hypothesized that the total smoking dose significantly affects LDH and that patients with a high smoking dose may have more severe symptoms and will require further surgical intervention.

MATERIALS AND METHODS

Subjects

In this study, 391 patients with LDH from our hospital were retrospectively evaluated. The LDH patients included in this study met the following three criteria:

a. LDH was the primary discharge diagnosis;
b. The patients were symptomatic and admitted for conservative or surgical treatment; and
c. There was a presence of radiological evidence of LDH between L1 and S1, on either a computed tomography scan (CT) or magnetic resonance imaging (MRI).

Another 431 patients from our hospital were enrolled as control subjects and retrospectively evaluated. These patients were randomly chosen according to every fifth medical record number from a medical or surgery department. Patients with a history of LDH were excluded. By assuming a smoking rate of 22% among the control subjects (the reported smoking prevalence in Taiwan),[12] a two-tailed significant level of 5%, and a power level of 90%, the enrollment of 391 case patients and 431 control subjects was expected to be adequate for detecting a minimal odds ratio (OR) of 2.[13] Surgical intervention was performed for more severe neurological deficits such as intolerable pain, progressive weakness of the lower legs, and bladder or rectum paresis, according to a previous report.[14] Patients with pediatric, obstetric, or psychiatric problems were also excluded. Information on discharge diagnosis, age, sex, body height, body weight, and level of cigarette consumption was obtained from a chart review. The smoking history with regard to the individual study subjects was obtained from the personal history of the attending physicians and also confirmed by a structured admission questionnaire provided by the initial caring nurse. The smoking behavior was classified into the following categories: (a) current or active smoker and (b) nonsmoker (<100 cigarettes over a lifetime and no regular smoking in the weeks before the baseline evaluation). *Smoking years* is defined as the number of years the patient smoked; *smoking dose*, is defined as the number of cigarette packs smoked per day (PPD) by the patient; and *total smoking dose* (pack-years) is defined as the smoking dose multiplied by the smoking years.

Subjects who had not changed their smoking behavior in the past years gave highly consistent responses on their smoking status (smokers or nonsmokers) and duration. The total smoking dose was employed to express the level of cigarette consumption.

Ethical considerations

This study was conducted in accordance with the Declaration of Helsinki 2000, with pertinent national and international regulatory requirements. The protocol was approved by the local ethics committee.

Statistical analysis

Descriptive data were assessed using the Student *t* test for continuous variables, and the chi-square test was utilized to evaluate the categorical variables in the demographic data. Multiple logistic regression analysis with stepwise selection was employed to assess the association of interests and to adjust for potential confounders. Statistical significance was set at $P < 0.05$. All the analyses were performed using the SPSS version 17.0 software (SPSS Inc., Chicago, IL, USA).

RESULTS

The key characteristics of the two study groups are presented in Table 1. Of the 822 patients, 567 (69.0%) were men. The mean ± SD age was 39 ± 18.4 years; body height -166.8 ± 9.1 cm; body weight - 66.9 ± 12.2 kg; and body mass index (BMI) - 24 ± 3.7. In total, 391 patients (47.6%) were assigned to the LDH group (292 men, 99 women); and 431

Table 1. Characteristics of the study population

Group	Study population		LDH		non-LDH		*P*
N	822		391		431		
Male, sex	567	69.0%	292	74.7%	275	63.8%	0.001
Age (years)	39.0	±18.4	35.5	±16.9	42.2	±19.1	<0.001
Body height (cm)	166.8	±9.1	168.5	±9.0	165.2	±9.0	<0.001
Body weight (kg)	66.9	±12.2	69.2	±12.6	64.9	±11.4	<0.001
BMI (kg / m^2)	24.0	±3.7	24.3	±3.7	23.8	±3.6	0.033
Smoking	294	35.8%	168	43.0%	126	29.2%	<0.001
PPD	0.30	±0.48	0.35	±0.47	0.25	±0.47	0.005
Pack-years*	3.8	±9.5	4.4	±10.1	3.4	±9.0	0.15
Years of smoking							<0.001
0 years	528	64.2%	223	57.0%	305	70.8%	
1-5 years	121	14.7%	76	19.4%	45	10.4%	
6-10years	81	9.9%	47	12.0%	34	7.9%	
>10 years	92	11.2%	45	11.5%	47	10.9%	
Surgery	264	32.1%	264	67.5%	0	0%	<0.001

Data are presented as mean and SD and numbers with percentages; Univariate continuous variables were assessed by student-t test and categorical variables were assessed by the chi-square test; LDH = lumbar disk herniation; PPD = packs smoked per day; *Years of smoking multiplied by PPD

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(52.4%) to the control group (275 men, 156 women). Among the patients, 341 (41.5%) were military personnel. In terms of smoking status, 294 patients (35.8%) were smokers with a mean smoking dose of 0.3 ± 0.48 PPD and mean total smoking dose of 3.8 ± 9.5 pack-years. The patients were classified according to the smoking years, as follows: 0 year (n = 528, 64.2%), 1-5 years (n = 121, 14.7%), 6-10 years (n = 81, 9.9%), and >10 years (n = 92, 11.2%). A total of 264 patients with LDH received surgical intervention (32.1% of the total patients and 67.5% of the patients with LDH).

Before adjusting for each variable, the patients in the LDH group were significantly younger than the patients in the control group (mean age, 35.5 ± 16.9 vs. 42.2 ± 19.2 years; P = 0.001). There were more men in the LDH group (n = 292, 74.7%) than in the control group (n = 275, 63.8%; P = 0.001). The mean body height in the LDH group was significantly higher than that in the control group (168.5 ± 9.0 cm vs. 165.2 ± 9.0 cm; P = 0.0001). The mean body weight in the LDH group was significantly heavier than that in the control group (69.2 ± 12.6 kg vs. 64.9 ± 11.4 kg; P = 0.0001). The mean BMI in the LDH group was significantly higher than that in the control group (24.3 ± 3.7 vs. 23.8 ± 3.6; P = 0.033). There were more military personnel in the LDH group (n = 205, 52.4%) than in the control group (n = 136, 31.6%; P < 0.0001). Cigarette smoking habits differed markedly between the LDH patients (168 / 391, 43.0%) and the controls (126 / 431, 29.2%; P < 0.0001). The mean number of cigarette PPD was higher in the LDH group than in the control group (0.35 ± 0.47 PPD vs. 0.25 ± 0.47 PPD; P = 0.005). The difference in smoking years was significant between the LDH and non-LDH groups (P = 0.0001). The proportion of smoking years for the 1-5 year, 6-10 year, and >10 year groups was higher in the LDH group (19.4, 12.0, and 11.5%, respectively) than in the non-LDH group (10.4, 7.9, and 10.9%, respectively).

The risk factors in the LDH group are presented in Table 2. The following 10 risk factors correlated significantly with LDH: Younger age (OR = 0.98; P < 0.001), increased height (OR = 1.04; P < 0.001), increased weight (OR = 1.03; P < 0.001), higher BMI (OR = 1.04; P = 0.03), more PPD (OR = 1.52; P = 0.005), male sex (OR = 1.67; P < 0.001), being military personnel (OR = 2.39; P < 0.001), smoking (OR = 1.82; P < 0.001), 1-5-year smoking history (OR = 2.31; P < 0.001), and 6-10-year smoking history (OR = 1.89; P = 0.008).

The risk factors for undergoing lumbar surgical intervention among the LDH group are presented in Table 3. The following seven risk factors correlated significantly with the need for lumbar surgical intervention for LDH: Increased weight (OR = 1.02; P = 0.006), higher BMI (OR = 1.06; P = 0.002), smoking (OR = 1.63; P = 0.001), more PPD (OR = 1.39; P = 0.03), more pack-years (OR = 1.02; P = 0.03), 6-10-year smoking history (OR = 1.84; P = 0.01), and >10-year smoking history (OR = 1.96; P = 0.004).

To further clarify the smoking-related risk factors associated with LDH and the need for lumbar surgical intervention, multiple logistic regression analysis was performed, to adjust for each variable in the independent samples, in Table 4. After adjusting for sex, age, body height, body weight, and BMI, pack-years, smoking, and a >10-year smoking history were found to be risk factors correlated with LDH (OR = 1.02, P = 0.02; OR = 1.51, P = 0.01; and

Table 2. Risk of LDH and associated factors in logistic regressions

Parameter	Estimate	SE	Odds ratio	95% CI		P-value
Age (years)	-0.02	0.004	0.98	0.97	0.99	<0.001
Body height (cm)	0.04	0.008	1.04	1.03	1.06	<0.001
Body weight (kg)	0.03	0.006	1.03	1.02	1.04	<0.001
BMI (kg/m^2)	0.04	0.02	1.04	1.00	1.08	0.03
Gender, Male vs. Female	0.51	0.15	1.67	1.24	2.26	<0.001
Smoking	0.60	0.15	1.82	1.37	2.43	<0.001
PPD	0.42	0.15	1.52	1.13	2.04	0.005
Pack-years*	0.01	0.007	1.01	1.00	1.03	0.15
Years of smoking						
0	—	—	1.00	—	—	—
1-5 years	0.84	0.21	2.31	1.54	3.47	<0.001
6-10years	0.64	0.24	1.89	1.18	3.04	0.008
>10 years	0.27	0.23	1.31	0.84	2.04	0.23

PPD = packs smoked per day; *Years of smoking multiplied by PPD

Table 3. Risk of lumbar surgery intervention and associated factors in logistic regressions

Parameter	Estimate	SE	Odds ratio	95% CI		P-value
Age	-0.002	0.004	0.99	0.99	1.01	0.63
Height	0.002	0.008	1.00	0.99	1.02	0.79
Weight	0.02	0.006	1.02	1.01	1.03	0.006
BMI	0.06	0.02	1.06	1.02	1.11	0.002
Gender, Male vs. Female	0.05	0.16	1.05	0.77	1.44	0.76
Smoking, Yes vs. No	0.60	0.15	1.63	1.21	2.21	0.001
PPD	0.33	0.15	1.39	1.03	1.88	0.03
Pack-years*	0.02	0.007	1.02	1.00	1.03	0.03
Years of smoking						
0 years	—	—	1.00	—	—	—
1-5 years	0.27	0.22	1.30	0.86	1.99	0.22
6-10 years	0.61	0.25	1.84	1.14	2.98	0.01
>10 years	0.67	0.23	1.96	1.24	3.08	0.004

PPD = packs smoked per day; *Years of smoking multiplied by PPD

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Table 4. Multiple logistic regression analyses for risk factors of LDH and lumbar surgery on smoking-related variables

Parameter	Univariate analysis				Multivariate analysis			
	Odds ratio	95% CI		*P*-value	Odds ratio[a]	95% CI		*P*-value
Lumbar HIVD								
Smoker Yes vs. No	1.82	1.37	2.43	<0.0001	1.51	1.09	2.10	0.01
PPD	1.52	1.13	2.04	0.005	1.30	0.94	1.78	0.11
Pack-years*	1.01	1.00	1.03	0.15	1.02	1.00	1.04	0.02
Years of smoking								
0	1.00	—	—	—	1.00	—	—	—
1-5 years	2.31	1.54	3.47	<0.0001	1.45	0.91	2.30	0.12
6-10years	1.89	1.18	3.04	0.008	1.44	0.86	2.40	0.16
>10 years	1.31	0.84	2.04	0.23	1.65	1.01	2.70	0.04
Lumbar surgery								
Smoker Yes vs. No	1.63	1.21	2.21	0.001	1.76	1.26	2.47	0.001
PPD	1.39	1.03	1.88	0.03	1.41	1.02	1.94	0.04
Pack-years*	1.02	1.00	1.03	0.03	1.02	1.00	1.03	0.03
Years of smoking								
0	1.00	—	—	—	1.00	—	—	—
1-5 years	1.30	0.86	1.99	0.22	1.39	0.86	2.26	0.18
6-10years	1.84	1.14	2.98	0.01	1.83	1.09	3.06	0.02
>10 years	1.96	1.24	3.08	0.004	2.16	1.31	3.54	0.002

[a]After adjusting for sex, age, height, body weight, and BMI; *Years of smoking multiplied by PPD

OR = 1.65, P = 0.04, respectively). In analyzing the risks for undergoing surgical intervention among the LDH group, PPD, pack-years, smoking, and a 6-10- or >10-year smoking history were found to be risk factors associated with the need for lumbar surgical intervention (OR = 1.41, P = 0.04; OR = 1.02, P = 0.03; OR = 1.76, P = 0.001; OR = 1.83, P = 0.02; OR = 2.16, P = 0.002).

DISCUSSION

Concurrent with the previous findings, the present results demonstrated that smoking, more pack-years, and a >10-year smoking history were the independent risk factors for LDH (OR = 1.51, P = 0.01; OR = 1.02, P = 0.02; and OR = 1.65, P = 0.04, respectively). Moreover, the results indicate that smoking dose, pack-years, and a >six-year smoking history may independently predict the need for surgical intervention in patients with LDH, which means that the more the pack-years and the longer the patient smoked, the greater the patient's potential for surgery. Therefore, smoking increases the need for surgical intervention for LDH in a dose- and time-dependent manner. To our knowledge, this is the first report of its kind.

Although controversial, the positive relationship between smoking and lower back pain and other disk disorders has been documented in many studies.[15,16] An *et al*.[17] demonstrated the positive association between smoking and LDH. Likewise, Kelsey *et al*.[18] reported the positive association between smoking and LDH, probably resulting from the increased intervertebral pressure caused by movement during smoke-induced coughing. Heliovaara *et al*.[19] found that smoking was the risk factor for LDH in men. Smoking was also demonstrated to be related to recurrent LDH after lumbar surgery.[20,21] On the contrary, another study did not indicate any significant relationship between being a smoker and being referred to a hospital for LDH.[22] All the subjects in that study were women. A previous report suggested that the risk of LDH among men was higher than that among women.[23] Men smoked nearly five times more than women worldwide.[24] This difference in sex ratio among the smoking population could be an important reason that accounts for the discrepancy in results. Some studies revealed no significant association between smoking and LDH or the risk of undergoing surgery for LDH.[25,26] None of these studies evaluated the pack-years and number of PPD. A multicenter case–control study in Germany revealed that a moderate number of pack-years (20 to 40 pack-years) was associated with LDH, but reported no clear relationship between a very high smoking dose (at least 40 pack-years) and LDH.[27] The small number of women who had consumed ≥40 pack-years could be the reason. Another systematic review

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showed no association between smoking and the need for lumbar surgical intervention.[28] However, these studies did not evaluate the pack-years and PPD. Overall, the previous reports revealed inconsistent results and lack of clear evidence of an association between the total smoking dose and LDH, and even the need for a lumbar surgery. The lack of data on the total smoking dose (pack-years) could be the reason for the inconsistency. Meanwhile, our results provide evidence of the presence of a positive association between LDH and the total smoking dose. Furthermore, the association between the need for lumbar surgical intervention and total smoking dose was demonstrated.

From a pathophysiological perspective, smoking could cause the lumbar disk to be more vulnerable to degenerative disk diseases, subsequently causing a prolapsed disk. The flexion and rotational forces on the annulus and even inherited factors have been known to lead to the development of LDH.[29] The association between smoking and LDH could be attributed to the following three mechanisms: First, frequent coughing caused by smoking could increase pressure on the disk.[30] Second, a study on rabbits revealed that nicotine affects the intervertebral disks by inducing a reduction in the density of vascular buds and a decrease in the vascular lumen, as a result of decreased oxygen tension, leading to a decreased synthesis of proteoglycan and collagen, thus facilitating disk degeneration.[31] As a degenerated disk is vulnerable, a radial fissure in the disk is easily noted. A radial fissure and subsequent gradual leaking of the nuclear pulposus into the spinal canal are probable causes of a prolapsed disk.[9] This hypothesis is the same as that postulated by Jensen *et al*.,[32] which states that disk degeneration is the basis for development of a prolapsed disk. Third, Ernst has reported that an insufficiency of oxygen and nutrients can cause the intervertebral disks to become more vulnerable to insults.[33]

The examination of dose-response relationships in previous studies revealed that increased cigarette consumption is related to the severity of back pain.[6] Smoking may affect the metabolism of the intervertebral disk and accelerate disk degeneration,[31] and act via the neuroendocrine system to alter pain perception, presumably increasing the back pain.[1] Smoking may also accelerate bone loss, thus leading to changes in the bone microarchitecture. Such changes may result in vertebral deformities and loss of spinal stability.[1] A more vulnerable disk, severe pain, and an unstable spine result in more severe symptoms and a need for further surgical intervention in patients with a higher smoking dose and longer smoking duration. Our study demonstrated that the smoking dose duration increased the need for surgical intervention, for LDH.

Moreover, increased weight and BMI were associated with LDH, which was concurrent with the results previously reported by Heliovaara,[34] who indicated that an increased BMI augmented the risk of LDH. Furthermore, this study demonstrated that an increased BMI was associated with a higher risk of undergoing lumbar surgical intervention for LDH. The present result was compatible with that reported by Rihn *et al*.,[35] which revealed that non-operative treatment was less clinically beneficial for obese patients, probably because of greater pressure exerted on the disk as a result of heavier weight, which exacerbated the symptoms.

Limitations

In this study, owing to the small sample size, the smokers were divided into two groups only: Current smokers and nonsmokers. Another limitation was that occupational risks were not classified because of chart recording limitations. Furthermore, smoking history was obtained from the retrospective data of chart records. The major disadvantage of using secondary data is that there is no way to know exactly how and how well the data were collected. To overcome such a drawback, the information (e.g., smoking history) was checked from both the personal history of the attending physicians and the structured admission questionnaire provided by the nurses, to reduce potential bias.

CONCLUSION

In conclusion, the results of the present study suggest that the total cigarette smoking dose is an independent risk factor for LDH. Furthermore, an increased total smoking dose is an independent risk factor for undergoing surgical intervention for LDH. Therefore, for maximal functional improvement, smoking should be avoided by patients undergoing treatment for LDH. Further research on the effect of smoking on LDH must be conducted in the future. Meanwhile, the treatment regimen and clinical benefit of quitting smoking require further study.

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