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Posted Date: 26 November 2024

doi: 10.20944/preprints202411.1872.v1

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Article

# The Relationship Between the Chronic Low Back Pain and Cigarette Smoking Habits in Patients Treated with Conservative Therapy

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**Abstract:** **Aim of the study;** this paper focus on the relationship between nicotine use and chronic lumbar back pain (CLBP). Any confirmation of the relationship between cigarette smoking and CLBP may help to educate patients on a healthy lifestyle for the prevention of the CLBP. **Materials and Methods;** This study is a cross-sectional observational study involving smokers and non-smokers patients with diagnosis of chronic low back pain (CLBP), recruited during their initial neurosurgical consultation at the Neurosurgery Clinic in a time period of 6 months. All patients were followed for a minimum of 3 months after the start of conservative therapy. For each patient were collected age and gender, smoking habits, and the presence of any comorbidities. Pain severity and discomfort were evaluated using the Numeric Rating Scale (NRS), the DN4 questionnaire and the the Oswestry Disability Index (ODI). Based on imaging (MRI of the lumbosacral spine), the diagnosis of disc herniation or vertebral lumbar stenosis was documented. Statistical analysis was performed using IBM SPSS Statistics software. A p-value of less than 0.05 was considered statistically significant. **Results;** in this study the improvement of the CLBP after the same conservative therapy as documented by NRS and DN4 scales is better in patients with non smoking habits respect to patients with smoking habit (NRS scale 0.001 and DN4 scale 0.027). **Conclusions** the cigarette smoking habit have a rule in the onset and maintenance of the chronic lumbar back pain in patients with spinal degenerative disease.

**Keywords:** low back pain; smoking and pain; chronic low back pain; spine fusion; spinal disc disease

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## 1. Introduction

The chronic low back pain (CLBP) is a common symptom in adult population and during their lifetime, the 80-90% of people experience at least one episode of low back pain [1,2].

The low back pain is classified as chronic when it persists for almost 3 months, at which point it becomes a pathology rather than a symptom [3]. The risk factors for the onset of LBP are many, such as: hereditary factors, age, gender, trauma, psychic factors (stress, anxiety, depression), heavy works and finally unhealthy lifestyle such as cigarette smoking and alcohol abuse.

Cigarette smoking has recently been studied because it is an unhealthy lifestyle that plays an important role in the musculoskeletal degeneration and in the onset of LBP. Nowadays in Italy the 24.2% of population smokes a percentage which increases every year especially among young population. Each cigarette introduces over 4,000 chemicals, at least eighty of them are carcinogenic according to the International Agency for Research on Cancer (IARC) [4].

It is well known that cigarette smoking acts as pro-inflammatory factor, establishing a pathophysiological link between smoking and chronic low back pain. In current literature the association between smoking and LBP is well documented. The mechanism of this association is unclear, potentially related to the cytotoxic effects of nicotine or other substances released by tobacco combustion. Additionally, cigarette smoking causes chronic inflammatory states, with high plasma

C-reactive protein (CRP) levels [5]. It is known that smoking reduces perfusion in the intervertebral disc, leading to malnutrition and degeneration.

Smoking is associated with headaches, trunk pain and pain in the extremities; the pain related with the smoking habits is poorly responsive to pharmacological therapy [5–7] Smokers not only have an increased risk, but also a slower recovery rate and a higher likelihood of recurrent hernias compared to non-smokers [7].

The goals of this study are to determine whether cigarette smoking is a risk factor for the onset of chronic low back pain and the different response to conservative therapy in smoker and non-smoker patients.

## 2. Materials and Methods

This study is a cross-sectional observational study involving patients with diagnosis of chronic low back pain (CLBP), recruited during their initial neurosurgical consultation at the Neurosurgery Clinic. All patients underwent conservative-medical treatment (desametasone 4mg /os for 1 week with paracetamol 1000 mg X2/day with bed rest, followed by ketoprofene 50 mg os for 10 days and paracetamole 1000 mg as needed, gabapentinoids 75 mg x2/day for one month; all the patients underwent to postural exercises/physiotherapy for 1 month).

The study involved patients who underwent conservative treatment at our Institution between January 2023 and June 2023; all patients were followed for a minimum of 3 months after the start of conservative therapy.

For each patient, demographic data were collected, including age and gender, smoking habits, and the presence of any comorbidities. Smoking status was evaluated by asking: do you currently smoke? Based on imaging (MRI of the lumbosacral spine), the presence of disc herniation and lumbar stenosis was evaluated.

Clinical data collected for each patient included: Pain severity using the Numeric Rating Scale (NRS), the presence of neuropathic pain using the DN4 questionnaire and the degree of disability using the Oswestry Disability Index (ODI).

These scales were assessed at the onset of symptoms and 3 months after the start of conservative treatment.

Statistical analysis was performed using IBM SPSS Statistics software. A p-value of less than 0.05 was considered statistically significant. Additionally, the means of the values obtained from the various scales and their respective standard deviations were calculated.

Any statistically significant differences in clinical conditions in LBP patients before and after therapy between smokers and non-smokers were reported.

## 3. Results

The cohort consists of 49 patients, of whom 31 were males (63%) and 18 were females (37%). Of these 49 patients, 25 were smokers (51%) while 24 were non-smokers (49%) (Table 1).

**Table 1.** General characteristics of patients (total patients num.=49).

Characteristics	Smokers (n=25)	Non-smokers (n=24)	p-value (<0.05)
Sex			0.218
male (n,%)	14 (28.6)	17 (34.7)	
female (n,%)	11 (22.4)	7 (14.3)	
Age (years, mean±DS°)	56 ±10.85	55 ±16.67	0.517
Comorbidities (num., mean±DS°)	0.64 ±0.65	0.45 ±0.64	0.970
NRS scale *			
onset (mean±DS°)	5.76 ±1.83	3.87 ±1.42	<b>0.001</b>
after 3-months of therapy (mean±DS°)	4.8 ±2.43	2.75 ±1.51	

DN4 scale $\infty$			
onset (mean $\pm$ DS $^\circ$ )	3.88 $\pm$ 1.12	3 $\pm$ 1.53	<b>0.027</b>
after 3-months of therapy (mean $\pm$ DS $^\circ$ )	2.72 $\pm$ 1.83	2.37 $\pm$ 1.53	
ODI scale $\times$			
onset (mean $\pm$ DS $^\circ$ )	34 $\pm$ 0.18	24 $\pm$ 0.13	0.096
after 3-months of therapy (mean $\pm$ DS $^\circ$ )	24 $\pm$ 0.20	18 $\pm$ 0.13	
Radiological data			
Disc herniation	20 (40.8)	21 (42.9)	
Lumbar stenosis	2 (4.0)	3 (6.1)	
Sine materia	3 (6.1)	0 (0)	

$^\circ$ DS: standard deviation.  $^*$ NRS scale: Numeric Pain Rating Scale.  $\infty$ DN4 scale: Douleur Neuropathique en 4 questions.  $\times$ ODI scale: Oswestry Disability Index.

The average age of the smokers was approximately 56 years  $\pm$  10.85, while the average age of the non-smokers was 55 years  $\pm$  16.67, with a range from 28 years for the youngest patient to 79 years for the oldest subject. Additionally, the anamnesis collected information on the presence of comorbidities, revealing that 27 patients (55%) did not have comorbidities, while the remaining 45% had arterial hypertension, diabetes mellitus, lipid metabolism disorders, rheumatologic disorders, and others. On average, smokers patients had 0.64 comorbidities with a standard deviation of  $\pm$  0.65, then the other had 0.45 comorbidities with a standard deviation of  $\pm$  0.64 (Table 1).

The average NRS scale value at the onset of symptoms in smokers was 5.76  $\pm$  1.83, while the average value in non-smokers was 3.87  $\pm$  1.42. The average of these values, calculated three months after conservative treatment, was 4.8  $\pm$  2.43 in smokers and 2.75  $\pm$  1.51 in non-smokers (Table 1).

The average DN4 questionnaire value at the onset in smokers was 3.88  $\pm$  1.12, while in non-smokers it was 3.00  $\pm$  1.53. Three months after treatment, the average DN4 value was 2.72  $\pm$  1.83 in smokers and 2.37  $\pm$  1.53 in non-smokers (Table 1).

The average ODI scale values at the onset of symptoms in smokers was 34%  $\pm$  0.18, while the average value in non-smokers at onset was 24%  $\pm$  0.13. The average ODI value after conservative treatment in smokers was 24%  $\pm$  0.20, while the average in non-smokers after treatment was 18%  $\pm$  0.13 (Table 1).

In our sample 41 patients (84%) had a characteristic radiological picture of disc herniation, 5 patients (10%) had lumbar stenosis, and 3 patients (6%) had spondyloarthritis without radiological evidence of disc herniation or lumbar stenosis (Table 1).

The statistical analysis provided the following results: using the Student's t-test, the age variable was studied in relation to smoking status, which was found to have a non-statistically significant difference ( $p=0.517$ ), as well as the analysis of the number of comorbidities related to smoking status, which also showed a non-statistically significant difference ( $p=0.970$ ). The analysis using the chi-square test of the relationship between gender and smoking status was not statistically significant ( $p=0.218$ ). The multiple linear regression between the various clinical scales (NRS, DN4, and ODI) at onset and after three months of conservative therapy, with the dependent variable (age, then number of comorbidities, and finally gender) was not statistically significant (Table 1).

Additionally, using the Student's t-test, the various clinical scales (NRS, DN4, and ODI) were analyzed, and the initial values compared between smokers and non-smokers were not statistically significant; for the NRS scale, the p-value was 0.472, for DN4 it was 0.204, and for ODI the p-value was 0.060. Finally, the values of the scales at onset were compared with those after three months of conservative therapy between smokers and non-smokers using multiple linear regression. It was found that the NRS values at onset compared to the NRS values three months after conservative therapy between smokers and non-smokers showed a statistically significant difference (p-value = 0.001). Similarly, the comparison of the DN4 questionnaire between onset and after three months of conservative therapy was statistically significant (p-value = 0.027). In contrast, the multiple linear

regression used for the comparison of the ODI scale values at onset and three months after therapy showed a non-statistically significant difference (p-value = 0.096) (Table 1).

#### 4. Discussion

This study reports the results of the conservative therapy on a population of 49 patients diagnosed with CLBP; in the total sample it was observed that the majority of patients with chronic lumbar pain were over 30 years old, which underscores literature findings that this condition's frequency increases with age [8].

Additionally, in this cohort of patient, there is a higher percentage of cigarette smoking CLBP patients (51%) compared to no-smoking group (49%), confirming various studies that consider cigarette smoking a risk factor for the onset of chronic lumbar pain [9].

As reported in literature this study confirm that there are differences in pain and functional recovery after conservative therapy in smoking and non-smoking patients. As regards age, number of comorbidities, and sex the improvement after therapy documented by the scales (NRS, DN4, ODI) in both groups were statistically non-significant. Therefore these variables can be considered non-interfering in our cohort.

The central focus of this study is the analysis of the initial clinical conditions of pain and the response to conservative therapy in patients with chronic lumbar pain, aiming to confirm that cigarette smoking patients at onset of the symptoms exhibit more severe pain compared to non-smoking and are associated with a worse prognosis. Our data shows that, at the clinical onset, smokers had a higher average NRS value (5.76) compared to non-smokers (3.87), indicating that smokers experienced more pain than non-smokers. This result was confirmed by Emanuel Schembri et al [9] in a wide number of patients. The average DN4 values at clinical onset were also slightly higher in smokers patients compared to non-smokers and the same for the ODI value. However, NRS scale and DN4 values did not show a statistically significant difference because at the onset of the disease the pain was experienced very strong in both group of patients.

The multiple linear regression analysis documented a statistically significant difference between the NRS and DN4 values for smokers and non-smokers, compared to the NRS and DN4 values three months after conservative treatment. In cigarette smoking patients the improvement after therapy was less relevant than non smoking patients that showed a rapid recovery with early return to daily activities and work. This highlights that, despite the therapeutic approach, cigarette smoking patients had a worse prognosis, respond less to therapies with bad outcome and persistence of pain.

The association between cigarette smoking and lumbar pain nowadays is well documented in literature and the possible causes discussed. Smuck et al [10] found that smoking is a risk factor for de novo low back pain [11–13] and neck pain [14]. The association of spine pain and head pain in cigarette smoking patients suggests an underlying vascular mechanism with the onset of vascular abnormalities [15,16]. The currently theory regarding the mechanism of the relationship between tobacco and disc degeneration hypothesize than tobacco is related to anoxia of the intervertebral disc consequent to vascular damage. Actually is well known that nicotine has direct negative effects on intervertebral discs, bone, and cartilage by lowering blood flow through vasoconstriction, reduction of the oxygen delivery to tissues with impaired metabolism and healing [17,18]. Elmasry et al. documented that nicotine reduces the proliferation rate and biosynthesis of glycosaminoglycan of disc cells, the reduction of exchange between blood vessels and disc tissue damage the nucleus pulposus with rapid degeneration and loss the physiological function [19]. Jakoi et al [20] documented that the association of cigarette smoking and obesity has a synergic effect on the damage of the lumbar disc. Yang QH et al [21] speculated that the frequent coughing in cigarette smoking patients may accelerate the process of disc degeneration cause the increase of the abdominal pressure with venous stagnation in the vertebral column plexus and damage of intervertebral disc.

In the advanced stage the damage of the intervertebral disc cause the disc herniation with mechanical compression of the nerve root and the release of many inflammatory mediators such as TNF, IL-1, IL-6, and IL-8 that play a pathophysiological role in the onset of low back pain and radicular pain [4,22–24].

Moreover it was documented that cigarette smoking increases the rate of non fusion in patients treated with spinal fusion surgery [25] with the late onset of pseudoarthrosis.

All these mechanism may explain the persistent LBP in cigarette smoking patients after proper pharmacological therapy as documented in our study.

Finally in this study the difference of ODI values at onset and after three months of therapy, when compared between smokers and non-smokers, were not statistically significant: this may be due because in most cases the chronic lumbar pain generally does not cause severe disability for the daily activities.

## 5. Conclusions

The study identified a series of significant findings regarding the association between the chronic lumbar pain and cigarette smoking. The results observed a higher percentage of smokers compared to non-smokers presenting with chronic lumbar pain. This not only confirms the association between tobacco use and the development of this condition but also it demonstrates that smoking can be considered a risk factor for the development of degenerative spinal diseases with chronic lumbar pain, in line with literature. Moreover, smoking can also be considered a negative predictive factor for the outcomes of patients after pharmacological therapy. This should motivate the scientific community to promote primary prevention in the population, and educate patients about an healthy lifestyle in order to limit harmful behavior to health. This study has some limiting factors such as the limited number of patients. Future studies will be necessary to confirm these results.

**Funding:** This publication was prepared without any external source of funding.

**Conflicts of Interest:** The authors declare no conflict of interest.

**Ethical approval:** Ethical approval was not necessary for the preparation of this article.

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