

Article

The Relationship Between 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 focuses on the correlation of nicotine use with chronic lumbar back pain (CLBP). **Methods:** This is a cross-sectional observational study involving smoker and non-smoker patients with a diagnosis of chronic low back pain (CLBP), recruited during their initial neurosurgical consultation at the Neurosurgery Clinic over a period of 6 months. All patients were followed for a minimum of 3 months after the start of conservative therapy. Age, sex, smoking habits, and the presence of any comorbidities were noted. Pain severity and discomfort were evaluated using the Numeric Rating Scale (NRS), the DN4 questionnaire, and the Oswestry Disability Index (ODI). Based on imaging (MRI of the lumbosacral spine), diagnoses of disc herniation or vertebral lumbar stenosis were documented. Statistical analysis was performed using IBM SPSS Statistics software (Version 30). A *p*-value of less than 0.05 was considered statistically significant. **Results:** Improvement on the CLBP, NRS, and DN4 scales after the same conservative therapy was better in Patients with non-smoking habits improved more on the CLBP, NRS and DN4 scales when compared to smokers (NRS scale 0.001 and DN4 scale 0.027). **Conclusions:** Patients with smoking habitudes affected by lumbar disk her-niation and stenosis and undergoing conservative therapy had worse pain scores

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



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1. Introduction

Chronic low back pain (CLBP) is a common symptom in the adult population. In total, 80–90% of people experience at least one episode of low back pain [1–3].

Low back pain is synthetically defined as chronic when it persists for almost 3 months [4]. Risk factors for the onset of LBP include heredity, age, gender, trauma, psychic factors (stress, anxiety, and depression), heavy work, and unhealthy lifestyles such as cigarette smoking and alcohol abuse [5].

Cigarette smoking, for a long time, has been studied because it is an unhealthy lifestyle that plays an important role in musculoskeletal degeneration and the onset of LBP [6]. In Italy, 24.2% of the population smokes, a percentage which increases every year, especially among the young population. Each cigarette introduces over 4000 chemicals, and at least 80 of them are carcinogenic according to the Italian Association for Cancer Research (IARC) [7].

It is well known that cigarette smoking acts as a pro-inflammatory factor, suggesting a pathophysiological link between smoking and chronic low back pain [8–10]. In the

current literature, the association between smoking and LBP is well documented [11–15]. 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 [16–19].

Beyond neoplasia, cigarette smoking impacts all phases of atherosclerosis, from endothelial dysfunction to acute vascular clinical events [19]. Several studies demonstrate that smokers exhibit reduced production of nitric oxide (NO), which not only regulates vasodilation, but also helps modulate inflammation, leukocyte adhesion, platelet activation, and thrombosis [17,18]. A study published in the American Journal of Medicine posits that tobacco smoking is a risk factor for the development of radicular pain, although its precise etiology remains unknown. It is hypothesized that smoking may reduce perfusion around the intervertebral disc, leading to malnutrition of these structures. Furthermore, not only do smokers have an increased risk, but they also exhibit a slower recovery rate and a higher probability of developing recurrent disc herniations compared to non-smokers. Additionally, the molecular mechanism of this might also be linked to the cytotoxic effects of nicotine or other substances released during tobacco combustion [20–22].

Smoking is also associated with migraines, truncal pain, and extremity pain, as well as fibromyalgia. There is also evidence that smoking may exacerbate migraine-related consequences (e.g., stroke) and lead to an increase in the systemic immunoinflammatory index in patients with fibromyalgia [23,24]. Pain in patients with smoking habit is poorly responsive to pharmacological treatment. Oral NSAIDs can be part of the management of low back pain, taking into account potential differences in gastrointestinal, hepatic, and cardio-renal toxicity, as well as individual patient risk factors, including age. Oral NSAIDs for low back pain should be prescribed at the lowest effective dose and for the shortest possible duration. Weak opioids, with or without paracetamol, should only be considered for the management of acute low back pain if NSAIDs are contraindicated, not tolerated, or have proven ineffective. Paracetamol alone should not be offered for the management of low back pain. Routine offering of opioids for acute low back pain is not recommended. Opioids should not be offered for the management of chronic low back pain [25]. Guided physical therapy has been explored as an alternative treatment to medical therapy. Sessions took place twice a week and lasted 45 min each. The intensity of the exercise was moderate. The program included strength, endurance, stretching, balance, and stabilization exercises. The subjects performed bodyweight resistance exercises and exercises with resistance. The aim of the resistance training was to strengthen some specific muscle groups, such as the erector spinae muscles and abdominis muscle. During resistance training, subjects performed isometric, concentric, and eccentric muscle work. The stretching exercises primarily involved the cervical and dorsal muscles and abdominal, ischiocrural and piriformis muscles. Each standard session of general rehabilitation gymnastics included a 10-min warm-up to prepare the muscles for proper training work [15,25–27]. Smokers not only have an increased risk, but also a slower recovery rate and a higher likelihood of recurrent hernias compared to non-smokers [26].

The goal of this study is to assess the different responses to conservative therapy in smoker and non-smoker patients.

2. Materials and Methods

This is a cross-sectional observational study involving patients with a diagnosis of chronic low back pain (CLBP), recruited during their initial neurosurgical consultation at the Neurosurgery Clinic.

The study was conducted in accordance with the Declaration of Helsinki. Ethical review and approval were waived as it was a retrospective study utilizing anonymized patient data. Informed consent was obtained from all subjects involved in the study.

Patients included in the study presented with chronic low back pain for more than 3 months, had not undergone chronic conservative therapy, and had a recent (maximum 1 month old) lumbosacral spinal MRI without contrast. Conversely, patients with low back pain for less than 3 months, those with paretic deficits of the lower limbs, those with a self-reported history of being an ex-smoker at the baseline visit or e-cigarette users, patients suffering from conditions mimicking the pain (e.g., affected by renal colic or peripheral obliterative arteriopathy), and those who did not fully adhere to the proposed therapy were excluded.

All patients underwent conservative medical treatment (dexamethasone 4 mg/os for 1 week with paracetamol 1000 mg twice a day with bed rest (prescribed for 4 days, with mobilization permitted for essential physiological needs), followed by ketoprofen 50 mg once a day for 10 days and paracetamol 1000 mg as needed (a maximum of 3 times per day), gabapentin 75 mg twice a day for one month; all patients underwent postural exercises/physiotherapy for 1 month).

This 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 sex, 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.

The 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, version 30 using the Student's *t*-test. 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 the clinical condition of LBP patients before and after therapy between smokers and non-smokers were reported.

3. Results

The cohort consisted of 49 patients, of whom 31 were males (63%) and 18 were females (37%). Of these 49 patients, 25 were smokers (51%, heavy smokers, identified as individuals consuming ≥ 1 pack/day for ≥ 1 year), while 24 were non-smokers (49%) (Table 1).

Among the 49 patients studied, the prevalence of current smokers was 51.02% ($n = 25$) (95% CI: 37.03–65.01%); however, the prevalence of non-smokers within the sample was 48.98% (95% CI: 34.99–62.97%).

The average age of smokers was 56 years $\pm$ 10.85 (ranging from 28 years for the youngest patient to 79 years for the oldest subject); that of non-smokers was 55 years $\pm$ 16.67 (ranging from 29 years for the youngest to 76 for the oldest). Additionally, medical records 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, and rheumatologic disorders. Smoker patients had

0.64 comorbidities with a standard deviation of ± 0.65 ; non-smokers had 0.45 comorbidities with a standard deviation of ± 0.64 (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 $\pm$ DS °)	56 $\pm$ 10.85	55 $\pm$ 16.67	0.517
Comorbidities (num., mean $\pm$ DS °)	0.64 $\pm$ 0.65	0.45 $\pm$ 0.64	0.970
NRS scale *			
Onset (mean $\pm$ DS °)	5.76 $\pm$ 1.83	3.87 $\pm$ 1.42	0.001
After 3 months of therapy (mean $\pm$ DS °)	4.8 $\pm$ 2.43	2.75 $\pm$ 1.51	
DN4 scale ∞			
Onset (mean $\pm$ DS °)	3.88 $\pm$ 1.12	3 $\pm$ 1.53	0.027
After 3 months of therapy (mean $\pm$ DS °)	2.72 $\pm$ 1.83	2.37 $\pm$ 1.53	
ODI scale ×			
Onset (mean $\pm$ DS °)	34 $\pm$ 0.18	24 $\pm$ 0.13	0.096
After 3 months of therapy (mean $\pm$ DS °)	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)	

° DS: standard deviation. * NRS scale: Numeric Pain Rating Scale. ∞ DN4 scale: Douleur Neuropathique en 4 questions. × ODI scale: Oswestry Disability Index.

The average NRS scale value at the onset of symptoms was 5.76 ± 1.83 in smokers and 3.87 ± 1.42 in non-smokers. NRS values three months after conservative treatment were 4.8 ± 2.43 in smokers and 2.75 ± 1.51 in non-smokers (Table 1).

The DN4 questionnaire values at onset were 3.88 ± 1.12 in smokers and 3.00 ± 1.53 in non-smokers. Three months after treatment, the DN4 value was 2.72 ± 1.83 in smokers and 2.37 ± 1.53 , in non-smokers (Table 1).

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

Forty-one patients (84%) had a localized displacement of disc material beyond the margins of the intervertebral disc space, while five patients (10%) had narrowing of the lumbar vertebra in the central canal, lateral recess, or neural foraminal areas. Three patients (6%) had central vertebral osteophytes but no radiological evidence of disc herniation or lumbar stenosis (Table 1).

Student's *t* test revealed no significant difference in age ($p = 0.517$) or number of comorbidities related to smoking status ($p = 0.970$). Chi-square analysis revealed no sex-related variation ($p = 0.218$). Multiple linear regression between the various clinical scales (NRS, DN4, and ODI) at onset and after three months of conservative therapy, considering the dependent variables (age, number of comorbidities, and sex), revealed no significant differences (Table 1).

The initial values compared between smokers and non-smokers were not statistically significant for the NRS scale ($p = 0.472$), DN4 ($p = 0.204$), or ODI ($p = 0.060$). NRS at onset compared to three months after conservative therapy between smokers and non-smokers showed a statistically significant difference ($p = 0.001$). Similarly, the comparison of the DN4 questionnaire between onset and after three months of conservative therapy was statistically significant ($p = 0.027$). In contrast, the multiple linear regression used for the comparison of the ODI scale values at onset and three months after therapy was not statistically significant ($p = 0.096$) (Table 1).

4. Discussion

This study reports the results of conservative therapy in a population of 49 patients diagnosed with CLBP.

As reported in the literature, this study confirms that there were differences in pain and functional recovery after conservative therapy in smoking and non-smoking patients [6,8–13,15]. Regarding age, number of comorbidities, and sex, the improvements after therapy documented by the scales (NRS, DN4, and ODI) in both groups were statistically non-significant. The number of patients enrolled, however, precludes excluding these potentially contributory factors.

The central aim of this study is to analyze initial pain and its response to conservative therapy in patients with chronic low back pain. Our data indicate that, at clinical onset, smokers presented a higher average NRS value (5.76) compared to non-smokers (3.87). This suggests that functional impairment at the onset of symptoms was already more pronounced in smokers than in non-smokers, with smokers reporting greater initial pain on a 1–10 scale compared to non-smokers. This finding was corroborated by Emanuel Schembri et al. [28] in a larger patient cohort. The average DN4 values at clinical onset were also slightly higher, but not statistically so, in smoker patients compared to non-smokers, with the same observed for the ODI value.

Multiple linear regression analysis documented 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. This highlights that, despite the therapeutic approach, cigarette smoking patients had a worse prognosis, responded less to therapies, and experienced bad outcomes and the persistence of pain, in terms of reported pain on a 1–10 numerical rating scale (NRS) and neuropathic pain (e.g., paresthesia) using the DN4 scale.

Today, the association between cigarette smoking and lumbar pain is well documented in the literature, and the possible causes have been discussed. Smuck et al. [29] found that smoking is a risk factor for de novo low back pain [30–32]. The association of spine pain and head pain in cigarette smoking patients suggests an underlying vascular mechanism with the onset of vascular abnormalities [33,34]. The current theory regarding the mechanism of the relationship between tobacco and disc degeneration hypothesizes that tobacco is related to anoxia of the intervertebral disc, consequent to vascular damage [8,12,16]. Nicotine affects intervertebral disc, bone and cartilage healing by reducing local blood flow and impairing metabolism [35,36]. Elmasry et al. documented reduced proliferation rate and glycosaminoglycan synthesis secondary to nicotine action, due to altered exchange between vessels and disc tissue and direct damage to the nucleus pulposus, leading to fast degeneration and function derangement [37]. Jakoi et al. [38] documented the association of cigarette smoking and obesity with damage of the lumbar disc. Yang QH et al. [5] speculated that smoking can increase the frequency of coughing, and coughing increases abdominal pressure, which intensifies the compression and stretch of the intervertebral disc on the nerve root, with venous stagnation in the vertebral column plexus, inflaming the nerve root and increasing edema and the sensitivity of the nerve to pain [5,39].

In the advanced stage, damage to the intervertebral disc causes 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, which may play a pathophysiological role in the onset of low back pain and radicular pain [40–42].

Moreover, it was documented that cigarette smoking increases the rate of non-fusion in patients treated with spinal fusion surgery [43] with a late onset of pseudoarthrosis.

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

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

It is important not to overlook certain criticalities that define the limitations of this study. Primarily, one must consider the small sample size. Bias could be attributed to the presence of comorbidities that might interfere with the final statistical results.

5. Conclusions

The study identified an association between chronic lumbar pain and cigarette smoking. It demonstrated that smoking can be considered a risk factor for the development of degenerative spinal diseases with chronic lumbar pain, in line with the literature. Moreover, smoking can also be considered a negative predictive factor for patient outcomes after pharmacological therapy. This study has some limiting factors, such as the limited number of patients. Future studies will be necessary to confirm these results. Future studies should motivate the scientific community to promote primary prevention in the population and educate patients about a healthy lifestyle to limit harmful behavior to health.

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: Data from this study is available after request to the authors.

Conflicts of Interest: The authors declare no conflicts of interest.

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