Submitted: 05 July, 2024 Accepted: 26 August, 2024 Published: 08 November, 2024 DOI:10.22514/sv.2024.144

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ORIGINAL RESEARCH

Investigating the effectiveness and safety of radiofrequency damage of the lumbar sympathetic ganglia on alcoholic peripheral neuropathy pain

Wei Zheng[1], Jiayu Lu[1], Wei He[1,*]

[1]Department of Anesthesiology, The Affiliated Hospital of Beihua University, 132011 Jilin, Jilin, China

***Correspondence**
whe2488@163.com
(Wei He)

Abstract

This study examines the effectiveness and safety of using radiofrequency damage to treat pain caused by alcoholic peripheral neuropathy by targeting the lumbar sympathetic ganglia. Alcoholic peripheral neuropathy is a form of peripheral neuropathy that occurs as a result of prolonged alcohol consumption. Radiofrequency damage of the lumbar sympathetic ganglia allows precise targeting and destruction of the lumbar sympathetic ganglia, frequently alleviating vascular or sympathetic reflex pain in the lower back and legs. A retrospective analysis was conducted on the clinical data of 100 patients who received treatment at our hospital from March 2021 to March 2023. Based on the treatment method, the patients were categorized into two groups: an experimental group (50 cases) and a control group (50 cases). The control group received conventional treatment, while the experimental group underwent radiofrequency damage to the lumbar sympathetic ganglia in addition to conventional treatment. The effectiveness and safety of the two treatment methodologies were evaluated and compared. After one month and three months of treatment, the experimental group demonstrated significantly lower pain scores and decreased levels of tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-8 (IL-8) as compared to the control group ($p < 0.001$). In addition, the experimental group showed faster motor nerve conduction velocity (MNCV) and sensory nerve conduction velocity (SNCV) in the median nerve and common peroneal nerve compared to the control group ($p < 0.05$). There was no statistically significant difference in the occurrence of complications between the two groups ($p > 0.05$). Radiofrequency damage of the lumbar sympathetic ganglion for treating pain caused by alcoholic peripheral neuropathy effectively inhibits inflammatory responses, significantly alleviates patient pain, improves nerve conduction function, and is safe, making it a promising clinical treatment.

Keywords

Radiofrequency damage; Lumbar sympathetic ganglia; Alcoholic peripheral neuropathy

1. Introduction

Prolonged and persistent alcohol intake can result in chronic alcoholism, which affects many parts of the brain, such as the peripheral nerves, cerebellum, cerebral cortex, brainstem and corpus callosum. This leads to the degeneration of tissues and irreversible neurological damage [1]. Alcoholic peripheral neuropathy is a prevalent form of peripheral neuropathy. The pathogenesis of alcoholic peripheral neuropathy remains a subject of controversy. In addition to the toxic effects of alcohol and its metabolites, other risk factors include malnutrition, vitamin B1 (thiamine) deficiency, and family history [2]. The main symptom of alcoholic peripheral neuropathy is pain, often accompanied by a burning sensation, which can be extremely unpleasant and potentially incapacitating. Patients may experience a greater impairment in their ability to perceive vibrations compared to their ability to sense position [3, 4].

This condition gradually worsens, causing weakness in the lower limbs. It first affects the distal extremities of the limbs and eventually results in the loss of reflexes in the ankles. As the condition advances, sensory and motor manifestations extend to the proximal limbs, culminating in diminished tendon reflexes across all four extremities, attenuated glove-and-stocking-type sensation, and ultimately, the development of an aberrant gait [5]. The elevated frequency of occurrence greatly affects patients' chances of survival and overall quality of life. Present clinical interventions strive to mitigate or arrest the advancement of diseases while also reversing and restoring pre-existing functional impairments. The primary interventions for this condition involve discontinuing alcohol

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consumption and providing vitamin B supplements. In more severe instances, rehabilitation therapy is also recommended [6].

Previous studies predominantly utilized pharmacological interventions, which yielded certain effectiveness. However, symptoms often recur after discontinuation. Radiofrequency damage of the lumbar sympathetic ganglia is a commonly employed clinical method for treating refractory pain. Further validation is necessary to determine whether the application of this treatment can accomplish its intended purpose in treating alcoholic peripheral neuropathy. This study aims to investigate the clinical efficacy of radiofrequency damage of the lumbar sympathetic ganglia in treating alcoholic peripheral neuropathy.

2. Materials and methods

2.1 General information

A retrospective analysis was conducted on the clinical data of 100 patients who received treatment for alcoholic peripheral neuropathy pain at our hospital from March 2021 to March 2023. Based on the treatment method, they were categorized into two groups: an experimental group of 50 cases and a control group of 50 cases. Table 1 shows the general information for both groups, demonstrating comparable baseline characteristics ($p > 0.05$).

Inclusion Criteria:

The following were the inclusion criteria.

(1) Patients who met the diagnostic criteria for alcoholic peripheral neuropathy: Diagnosed with alcoholic peripheral neuropathy through clinical symptoms, laboratory tests, medical history and electromyography, the patient exhibits sensory and motor impairments beginning in the extremities, particularly the lower limbs, which gradually progress proximally in a symmetrical manner. This is accompanied by moderate to severe numbness, tingling, and burning pain in the lower limbs, burning or numbness in the soles of the feet, a feeling of heat and cramping pain in the calf muscles [7].

(2) Patients with a history of alcohol consumption, with an intake exceeding 150 g/day.

(3) Patients who exhibited signs of peripheral nerve involvement.

(4) Patients who signed informed consent form.

Exclusion Criteria:

The following conditions were considered for the exclusion criteria.

(1) Patients with peripheral neuropathy not caused by alcohol.

(2) Patients with severe hepatic and renal dysfunction or cardiopulmonary insufficiency.

(3) Patients with accompanying neurological diseases or consciousness disorders.

(4) Patients with poor compliance or withdrawal from the study midway.

2.2 Methods

Patients in the control group received conventional treatment, which included intramuscular injections of Vitamin B1 (100 mg) and Vitamin B12 (500 mg), administered once every two days. Additionally, attending physicians and nurses provided psychological counseling and alcohol cessation education. Other vasodilators, analgesics, and neurotrophic drugs were discontinued during treatment.

The treatment in the experimental group involved inducing radiofrequency damage to the lumbar sympathetic ganglia, building upon the methods used in the control group. The procedure was as follows: the patient was positioned supine on the digital subtraction angiography (DSA) imaging table. The L2 or L4 vertebral body on the affected side was selected as the puncture site. Routine disinfection and draping were performed. A puncture needle was then inserted at an angle of 45° to 60° into the skin. Under C-arm fluoroscopy, the needle tip was advanced to the target vertebral body's facet joint line in the anteroposterior view, reaching slightly anterolaterally in the lateral view. Before administering the contrast agent, aspiration was performed using a syringe to ensure the absence of blood or gas. 2 mL of iopamidol (370) was injected. In the anteroposterior radiograph, the contrast agent diffused around the small joint line of the affected vertebral body, and in the lateral radiograph, it diffused longitudinally along the anterolateral aspect of the vertebral body. Through this channel, 2 mL of 1% lidocaine was injected. Subsequently, the radiofrequency electrode was inserted to perform continuous radiofrequency damage to the sympathetic nerve using a Baylis-230 radiofrequency device. The specific parameters for the radiofrequency thermocoagulation were as follows: The frequency was set to 50 Hz and the voltage to 1.0 V. After identifying the patient's pain points, the frequency was adjusted to 2 Hz and the voltage to 2.0 V in order to induce muscle twitching in the lower limbs. Finally, a trial block was conducted by injecting 5 mL of 1% lidocaine, and the patient's vital signs and reactions were closely monitored.

2.3 Observation indicators

(1) Pain Score and Inflammatory Cytokine Levels: Before and after one month and three months of post-treatment, the pain scores and levels of inflammatory cytokines were compared between the two groups. Pain scores was measured

TABLE 1. Comparison of general information between two groups ($\bar{x} \pm s$).

Group	n	Age (yr)	Gender (Male/Female)	Years of drinking alcohol (yr)
Experimental group	50	42.21 ± 9.16	32/18	11.50 ± 3.23
Control group	50	41.67 ± 9.10	30/20	12.20 ± 3.35
t	-	0.300	0.170	1.064
p	-	0.765	0.680	0.290

using the Visual Analog Scale (VAS) [8], with a total score ranging from 0 to 10; higher scores indicate stronger pain. Inflammatory cytokines include Tumor Necrosis Factor-alpha (TNF-α), Interleukin-6 (IL-6), and Interleukin-8 (IL-8).

(2) Neural Conduction Function: Neural conduction function was evaluated using the electromyograph from Danish company Dantec (Cantata^{TM}, Copenhagen, Denmark). The motor nerve conduction velocity (MNCV) and sensory nerve conduction velocity (SNCV) of the median and common peroneal nerve were measured before and after treatment for both groups.

(3) Complications: Instances of minor dizziness, dry mouth, constipation, anorexia and other complications were recorded during the treatment period.

2.4 Statistical methods

The data were analyzed using the SPSS 22.0 statistical software (IBM, Armonk, NY, USA). Measurement data were described as ($\bar{x} \pm s$) and compared using t-tests. The count data were expressed as percentages (%) and compared by χ^2 tests. A p-value less than 0.005 ($p < 0.05$) indicates statistically significant differences.

3. Results

3.1 Pain scores and inflammatory cytokine levels

At one and three months post-treatment, the experimental group exhibited significantly lower VAS scores, as well as decreased levels of TNF-α, IL-6 and IL-8, compared to the control group ($p < 0.05$) (Tables 2,3).

3.2 Neural conduction function

Post-treatment, the MNCV and SNCV of the median and common peroneal nerves in the experimental group were found to be faster compared to the control group ($p < 0.05$) (Tables 4,5).

3.3 Complications

The incidence of complications did not differ significantly between the two groups ($p > 0.05$) (Table 6).

4. Discussion

Alcoholic peripheral neuropathy, a prevalent nutritional complication, is directly associated with prolonged alcohol consumption. The incidence of this condition correlates significantly with both the duration and volume of alcohol consumed [9]. Clinically, it manifests as axonal length-dependent peripheral neuropathy with progressive sensory involvement. There is a wide variation in the degree of clinical symptoms among patients. The initial symptoms include abnormal sensation in the toes, which then spreads to the upper and lower limbs. This progression eventually leads to pain, numbness, weakness in the lower limbs, muscle atrophy in the distal limbs, impaired deep sensory perception, ataxia and autonomic nervous system dysfunction in the later stages [10].

The L2 ganglion of the lumbar sympathetic ganglia is essential in the pathophysiology of this condition. The treatment of peripheral neuropathy pain usually involves targeting the lumbar sympathetic ganglion at the L2 level to enhance local blood flow and nutrient delivery, decrease the occurrence of vascular spasms and ischemic pain, and ultimately alleviate symptoms of limb reflex sympathetic pain [11, 12].

In a clinical context, patients frequently exhibit symptoms including hypoesthesia, pain in the limbs and sensations of burning pain. Previously, lumbar sympathetic ganglion block was routinely utilized to alleviate the discomfort associated with alcoholic peripheral neuropathy. While this approach significantly relieves pain, its efficacy is sometimes short-lived, necessitating repeated punctures. This heightens the likelihood of complications associated with treatment and adversely affects the prognosis [13, 14].

As such, the necessity for an expedient and effective treatment approach has grown more pressing. The radiofrequency damage of the lumbar sympathetic ganglion represents a minimally invasive intervention. This procedure employs thermal energy to selectively destroy sections of the lumbar sympathetic nerve tissue, thereby disrupting neural functions. The treatment involves placing a puncture needle at the site of the sympathetic ganglia and using radiofrequency thermal coagulation to destroy some of the postganglionic fibers. This is done with the intention of achieving various therapeutic goals.

These actions include reducing sympathetic excitability, dilating lower limb blood vessels, enhancing arterial and venous blood flow in the lower limbs, improving ischemic conditions of the nerve roots, and establishing collateral circulation. The findings of this study indicated that one and three months following treatment, individuals in the experimental group exhibited significantly lower pain scores and a reduction in the levels of inflammatory cytokines (TNF-α, IL-6, IL-8) when compared to those in the control group. This indicates that radiofrequency damage of the lumbar sympathetic ganglia is more effective than conventional clinical treatment for alleviating pain associated with alcoholic peripheral neuropathy. Radiofrequency damage of the lumbar sympathetic ganglia is a minimally invasive therapeutic procedure that inflicts minimum damage, enabling patients to promptly return to their regular activities.

The lumbar sympathetic ganglion, particularly at the L2 level, is essential in controlling neuropathic pain in the lower limbs. Research indicates that the primary approach for managing lower limb neuropathic pain centers on the obstruction or dissection of the lumbar sympathetic ganglion at the L2 level. This strategy aims to enhance local blood circulation and nutrient delivery, diminish vascular spasms and ischemic discomfort, and consequently mitigate lower limb reflex sympathetic pain [15]. Lumbar sympathetic ganglion radiofrequency thermocoagulation is an accurate surgical procedure targeting a specific location and achieving controlled tissue damage [16]. The radiofrequency needle is precisely positioned at the afflicted site using the electrical stimulation function of a radiofrequency-controlled thermostatic coagulator. Precise ablation of the lesion can be achieved by adjusting the radiofrequency output power and operating temperature to the optimum levels. Empirical evidence demonstrates that elevating the temperature of the radiofrequency needle to 80 °C can lead

TABLE 2. Comparison of VAS scores ($\bar{x} \pm s$, points).

Group	n	Before treatment	After 1 month of treatment	After 3 months of treatment
Experimental group	50	7.50 ± 0.65	3.10 ± 1.15	2.86 ± 0.95
Control group	50	7.34 ± 0.82	4.22 ± 0.91	4.12 ± 0.94
t	-	1.081	5.408	6.675
p	-	0.283	<0.001	<0.001

TABLE 3. Comparison of inflammatory cytokine levels ($\bar{x} \pm s$, pg/mL).

Group	n	TNF-α		
		Before treatment	After 1 month of treatment	After 3 months of treatment
Experimental group	50	25.58 ± 1.33	17.60 ± 1.25	15.24 ± 1.30
Control group	50	25.61 ± 1.28	20.34 ± 1.32	17.65 ± 1.48
t	-	0.115	10.594	8.695
p	-	0.909	<0.001	<0.001
Group	n	IL-6		
		Before treatment	After 1 month of treatment	After 3 months of treatment
Experimental group	50	95.81 ± 1.05	48.46 ± 1.19	19.05 ± 1.18
Control group	50	95.48 ± 1.06	54.61 ± 1.30	22.19 ± 1.16
t	-	1.601	24.722	13.433
p	-	0.113	<0.001	<0.001
Group	n	IL-8		
		Before treatment	After 1 month of treatment	After 3 months of treatment
Experimental group	50	178.56 ± 1.47	111.54 ± 1.20	77.61 ± 1.13
Control group	50	178.62 ± 1.50	123.43 ± 1.34	90.50 ± 1.47
t	-	0.202	46.842	49.348
p	-	0.840	<0.001	<0.001

TNF-α: Tumor Necrosis Factor-alpha; IL-6: Interleukin-6; IL-8: Interleukin-8.

TABLE 4. Comparison of median nerve conduction function of the two groups ($\bar{x} \pm s$, m/s).

Group	n	MNCV		SNCV	
		Before treatment	After treatment	Before treatment	After treatment
Experimental group	50	42.25 ± 4.13	51.64 ± 5.50	39.67 ± 3.12	48.90 ± 4.62
Control group	50	42.06 ± 4.23	47.60 ± 4.71	40.05 ± 3.09	44.75 ± 3.72
t	-	0.227	3.944	0.599	4.950
p	-	0.821	<0.001	0.551	<0.001

MNCV: motor nerve conduction velocity; SNCV: sensory nerve conduction velocity.

TABLE 5. Comparison of the common peroneal nerve function of the two groups ($\bar{x} \pm s$, m/s).

Group	n	MNCV		SNCV	
		Before treatment	After treatment	Before treatment	After treatment
Experimental group	50	36.12 ± 6.80	42.21 ± 7.52	31.60 ± 10.78	40.45 ± 13.69
Control group	50	35.70 ± 7.21	37.45 ± 7.35	30.00 ± 9.35	34.10 ± 9.98
t	-	0.300	3.196	0.793	2.651
p	-	0.765	0.002	0.430	0.009

MNCV: motor nerve conduction velocity; SNCV: sensory nerve conduction velocity.

TABLE 6. Comparison of the complications (n (%)).

Group	n	Mild dizziness	Thirst	Astriction	Total
Experimental group	50	2 (4.00)	1 (2.00)	1 (2.00)	4 (8.00)
Control group	50	1 (2.00)	1 (2.00)	3 (6.00)	5 (10.00)
t	-				0.122
p	-				0.727

to the breakdown and death of the unmyelinated C fiber axons present in the patient's nerve fibers. This effectively obstructs their ability to transmit neural signals, resulting in a notable alleviation of symptoms associated with neuropathic pain [17].

Neural function impairment leads to the activation of inflammatory responses and the release of inflammatory cytokines, which harm nerve tissues [18]. TNF-α and IL-6 are cytokines secreted in the initial phases of inflammation. They facilitate the cascade activation of inflammatory responses by recruiting and activating various inflammatory cells, promoting the synthesis and secretion of multiple inflammatory mediators. Additionally, they possess toxic and demyelinating effects on neurons and glial cells, directly causing peripheral nerve damage [19, 20]. Radiofrequency damage of the lumbar sympathetic ganglia can partially impede inflammatory responses, hence mitigating neurological function impairment resulting from inflammation. By combining with lidocaine, it is able to achieve therapeutic goals without causing complete destruction of all sympathetic nerves, as it specifically targets and destroys local sympathetic nerve tissue [21].

As a result, the inflammatory response caused by lesions is effectively inhibited, levels of pain-inducing factors are greatly decreased, and improved pain relief is attained. The levels of inflammatory cytokines in patients decline even more post-treatment. In Huang Youqing's study, a total of 116 patients who had just been diagnosed with postherpetic neuralgia (PHN) were randomly assigned to either a control group or an observation group, with 58 patients in each group. The control group was administered oral medication, while the observation group received oral medication and pulsed radiofrequency damage to the dorsal root ganglion.

The results indicated that pain scores in the observation group were significantly lower than in the control group. Additionally, the blood levels of IL-6, C-reactive protein (CRP) and TNF-α in the observation group were significantly lower than in the control group [22]. These findings demonstrate the safety and effectiveness of ganglion radiofrequency damage for treating PHN. The efficacy of this medication may be attributed to enhanced T-cell immunity and its inhibition of inflammatory responses. This is consistent with the findings of our study, which indicate that ganglion radiofrequency damage can effectively alleviate pain caused by peripheral neuropathy. The experimental group had faster MNCV and SNCV of the median and peroneal nerves than the control group. This indicates that radiofrequency damage of the lumbar sympathetic ganglia for treating alcoholic peripheral neuropathy pain can enhance neural conduction speed and improve neural conduction function.

There are multiple factors that contribute to this improvement. One of them is the damage caused by lumbar sympathetic ganglion radiofrequency, which can improve blood circulation disorders, speed up the removal of substances that cause pain, facilitate the recovery of organ functions, enhance the body's ability to resist diseases and improve the capability to maintain internal environmental balance. Long-term disruption of lumbar sympathetic nerve function results in sustained vasodilation, enhancing the supply of blood and nutrients to tissues, eliminating aberrant sensations, and lowering pain. Furthermore, sympathetic nerves regulate vascular dilation and constriction, and their destruction can alleviate vascular spasms in the lower extremities, improve circulatory disorders, and reduce vasospastic or ischemic pain. Relieving pain also enhances blood circulation, restoring arterial pulsation in the feet, which is crucial for preserving the limbs [23, 24].

Pre-injection of lidocaine before radiofrequency therapy not only simulates the effectiveness of the treatment but also prevents complications such as burning pain resulting from local stimulation induced by radiofrequency and destructive drugs. This enhances patients' ability to heal at a faster rate and promotes the restoration of damaged nerve cells, hence expediting the restoration of neural transmission function. As a result, after the treatment, the motor and sensory nerve conduction velocities in the median and peroneal nerves within the treatment group showed a significant improvement. The difference in complication rates between the two groups was not statistically significant, suggesting that the use of radiofrequency damage for managing pain caused by alcoholic peripheral neuropathy is considered safe. Radiofrequency thermocoagulation does produce considerable scar tissue and has the advantage of repeatability. It has a high success rate in puncturing lumbar sympathetic ganglia and successfully ablating them while also decreasing the occurrence of complications with the aid of X-ray fluoroscopy [25].

This study has certain limitations, including a relatively small sample size and a lack of comprehensive examination of patients' baseline conditions and general information. The constrained sample size and the study's confinement to a single center may impede the broad applicability of its findings. Furthermore, the factors that influence postoperative complications have not been extensively investigated. Supplementary analyses are required to advance the improvement of surgical and clinical management practices. Subsequent investigations should strive to use more extensive sample sizes and a broader range of patient demographic information. Conducting multicenter studies would enhance the robustness and generalizability of the findings. Moreover, systematically examining risk factors linked to postoperative complications is essential for a comprehensive understanding. In order to improve the accuracy and comprehensiveness of study outcomes, future research should include data and discussions on patient recur-

rence.

5. Conclusions

The use of radiofrequency ablation targeting the lumbar sympathetic ganglia in the treatment of pain associated with alcoholic peripheral neuropathy has been found to effectively inhibit inflammatory responses and reduce the levels of pain-inducing factors. This intervention significantly alleviates pain and enhances neural conduction function, demonstrating a high safety profile. Its effectiveness and safety warrant broader consideration and adoption in clinical practice.

AVAILABILITY OF DATA AND MATERIALS

The authors declare that all data supporting the findings of this study are available within the paper and any raw data can be obtained from the corresponding author upon request.

AUTHOR CONTRIBUTIONS

WZ—designed the study and carried them out. WZ, JYL, WH—supervised the data collection. WZ, JYL—analyzed the data; interpreted the data. WZ, WH—prepared the manuscript for publication and reviewed the draft of the manuscript. All authors have read and approved the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was obtained from the Ethics Committee of the Affiliated Hospital of Beihua University (Approval no. 20240013). Written informed consent was obtained from a legally authorized representative for anonymized patient information to be published in this article.

ACKNOWLEDGMENT

Not applicable.

FUNDING

This work was supported by Science and technology research project of Jilin Education Department (Grant No. JJKH20210060KJ).

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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How to cite this article: Wei Zheng, Jiayu Lu, Wei He. Investigating the effectiveness and safety of radiofrequency damage of the lumbar sympathetic ganglia on alcoholic peripheral neuropathy pain. Signa Vitae. 2024; 20(11): 56-62. doi: 10.22514/sv.2024.144.