Case-Control Study

Functional Pathway Between Cervical Spinal and Sympathetic Ganglia: A Neurochemical Foundation Between Neck Pain and Vertigo

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Background: Cervical vertigo commonly concurs in patients with neck pain, but the concurrent mechanism of these 2 symptoms still remains unclear. We previously reported a bidirectional segmental nerve fiber connection between cervical spinal and sympathetic ganglia, which provided a hypothesis that this connection between the 2 ganglia may be the anatomic basis for the concurrence of neck pain and cervical vertigo. However, this concurrent mechanism needs biochemical and functional evidence.

Objectives: This study aimed to investigate a possible noradrenergic pathway between cervical spinal and sympathetic ganglia.

Study Design: We performed both clinical and laboratory research. Clinical observation was a prospective case–control study.

Setting: Clinical study took place in our hospital; laboratory study was in an orthopedic laboratory.

Methods: Cervical lamina block therapy used in patients with cervical vertigo was clinically evaluated; norepinephrine (NE) expressions in cervical sympathetic ganglia were analyzed using immunohistochemical staining after electrical stimulation to the cervical spinal ganglia; the influence of phentolamine local injection to the vertebrobasilar artery flow was experimentally measured.

Results: Cervical lamina block therapy could significantly shorten the clinical hospital stays of patients with cervical vertigo ($P = 0.000$) and improve vertebral artery flow ($P < 0.05$). NE expressions in superior cervical sympathetic ganglia (SCG) or inferior cervical sympathetic ganglia (ICG) increased significantly when ipsilateral C2 to C3 or C6 to C8 spinal ganglia were electrically stimulated, respectively. Adrenergic receptor block with phentolamine significantly inhibited the decrease of basilar artery (BA) flow induced by electrical stimulation of the cervical spinal ganglia. The change range of BA flow caused by stimulations of C2 to C3 and C6 to C8 spinal ganglia was more than that of C4 and C5.

Limitations: The inpatients observed in this clinical study might be influenced by some factors including emotion, diet, sleep, and others. The limitations of the laboratory study included animal species and small sample size.

Conclusions: Adrenergic system could play a part in cervical spinal ganglia altering the vertebrobasilar artery system. It could provide a neurochemical foundation between neck pain and vertigo, and that segmental functional connections exist between cervical spinal and sympathetic ganglia.

Key words: Cervical vertigo, neck pain, cervical sympathetic ganglia, cervical spinal ganglia, noradrenaline

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Cervical vertigo is an illusory motion deriving from a disturbance of the neck. Thus far, the pathogenesis of cervical vertigo remains controversial. There are different hypotheses to explain the pathogenesis of cervical vertigo, such as proprioceptive cervical vertigo, Barré-Liéou syndrome, rotational vertebral artery (VA) vertigo, and migraine-associated cervical vertigo (1). Each has a different pathogenesis mechanism and diagnostic characteristics. No matter which kind of cervical vertigo, neck pain and vertigo symptoms commonly concur simultaneously. We previously tried to explain if there is any possible connection between the simultaneous existence of the 2 symptoms in most cases of cervical vertigo, and reported bidirectional segmental nerve fiber connections between the cervical spinal and sympathetic ganglia, which may be used to explain the pathogenesis of cervical vertigo in patients with neck pain (2).

To validate the existence of such a connection, we performed a retrospective study of patients with vertigo treated with cervical lamina block therapy, which indicated that cervical lamina block therapy could promote the recovery of vertigo and improve vertebrobasilar artery insufficiency. Cervical lamina is mainly innervated by the spinal nerve (3,4), and the block therapy could prevent the neck signal transmission from the spinal nerve to the sympathetic nerve and brain. Therefore, neck pain and vertigo are simultaneously relieved. This clinical analysis supported our previous hypothesis that some connections exist between the cervical spinal and sympathetic ganglia. To further consolidate this opinion, we tried to identify any functional connection between the cervical spinal and sympathetic ganglia such as neurotransmitters. Norepinephrine (NE), the most important neurotransmitter of sympathetic postganglionic fibers, is one of the important elements to regulate vertebral arteries contraction and cerebral blood supply (5). Animal experiments indicated that electrical stimulation of the cervical spinal ganglia could increase the NE level in the cervical sympathetic ganglia and decrease basilar artery (BA) flow, and adrenergic receptor blockage could inhibit the electrical stimulation-induced vertebrobasilar artery flow changes. This report supported our previous hypothesis regarding the connections between the cervical spinal and sympathetic ganglia clinically and functionally and may provide a possible neurochemical foundation for the pathogenesis of cervical vertigo.

Methods

Clinical Study

Subjects

Ninety patients with vertigo and neck pain (mean age, 56.5 ± 3.1 years; mean body weight, 76.4 ± 8.3 kg; mean height, 175.6 ± 7.3 cm) were randomly divided into the control group (n = 42) and the experimental group (n = 48). Patients with other dizziness diseases of eye, ear, and brain were excluded. There was no significant difference in gender, age, and disease duration between patients in the control and experimental groups (Table 1).

Methods

Patients in the control group were treated with vasodilator, and patients in the experimental group were treated with cervical lamina block therapy besides vasodilator. The block therapy included Adcortyl 20 mg (Xianju Inc., Zhejiang, China), vitamin B1 0.1 g, vitamin B12 0.2 mg, and 2% lidocaine 5 mL. According to patients' condition, another block therapy could be conducted at 1 week after the first block therapy.

The Dizziness Assessment Rating Scale (DARS) and the Visual Analog Scale (VAS) were used to assess the changes of vertigo and neck pain symptoms. Color Doppler ultrasound (CDU) was used to measure bilateral VA flow changes.

Statistical Methods

One-way analysis of variance or t test was used for the comparison between the control and experimental

Table 1. *General characteristics of the patients.*

Groups	n	Gender		Age	Disease Duration (years)		
		Male	Female		< 2	2-5	> 5
Control group	42	19	23	55.7 ± 2.8	8	24	10
Experimental group	48	21	27	58.1 ± 3.3	9	28	11

groups. The data collected were processed using SPSS Version 19.0 software (IBM Corporation, Armonk, NY). Values of $P < 0.05$ was considered to be significant.

Animal Study

Animals and Experimental Groups

A total of 198 healthy New Zealand rabbits (weight range, 2.5-3.0 kg) were randomly divided into 7 experimental groups and 1 control group. According to the stimulated spinal ganglia C2 to C8, experimental groups were defined as C2 to C8 groups respectively (n = 27 in each group). Animals in each experimental group were randomly divided into normal, saline solution, and phentolamine (an adrenergic blocker) treatments (n = 9 in each treatment). Control group without stimulation and treatment were defined as C0 (n = 9). The use of experimental animals was approved and supervised by the institutional animal welfare and research committee.

Electrical Stimulation to the Cervical Spinal Ganglia

Anesthesia was induced by intravenous administration of chloral hydrate (60 mg/kg). Experimental rabbits were placed in the prone position on an insulated laboratory table. Spinal segments were marked by fluoroscopy. Corresponding facet joints were cut to expose the left C2 to C8 spinal ganglia, respectively. The spinal ganglia were insulated from surrounding soft tissue using paraffin oil. Electrode was penetrated into the spinal ganglia. Electric stimulus was conducted with intensity 10 V, frequency 30 Hz, and duration 5 minutes. Deep anesthesia was kept to avoid animal suffering during the experiments.

BA Flow Measurement

After the exposure of the spinal ganglia, 5 mL saline solution or 5 mg (5 mL) phentolamine was injected into the soft tissue surrounding the whole VA under Doppler ultrasound. BA flow was measured through the foramen magnum using CDU 15 minutes after the application of saline solution or phentolamine, then electrical stimulation to the cervical spinal ganglia was conducted. BA flow was measured again when electrical stimulation was conducted. In normal treatment, BA flow was measured at the same time interval, and BA flow changes were recorded.

Preparation of Slides

Rabbits were euthanized using rapid perfusion through the left ventricle with physiological saline solution at 37°C, then slow perfusion was conducted with 4% paraformaldehyde at room temperature for about 1 hour. Left cervical sympathetic ganglia were cut and postfixed in 4% paraformaldehyde solution for 6 hours immediately after perfusion, then embedded with optimal cutting temperature medium after completely submersed in sucrose solutions. Eight micrometer thick cryosections were cut and placed on the slides pretreated with Poly-L-lysine hydrobromide.

Immunohistochemical Staining and Analysis

Immunohistochemical staining of cryosections was carried out by a pathological technician. Noradrenaline antibody (ab8887, Abcam, Cambridge, UK) and Biotin-Streptavidin HRP Detection Systems (SP-9001, Zhongshan Goldenbridge Company, Beijing, China) were used in this study.

Each section was observed under electron microscopy (DS-Fi2, Nikon, Tokyo, Japan), pictures taken under 200 times and assayed using Image-Pro Plus 6.0 software (Media Cybernetics, Rockville, MD). The optical density values in 5 pictures on the same section were taken as the mean optical density (MOD) of this section. The data were presented as $\overline{X}$ ± standard deviation.

Data Analysis

All data of the experimental group and control group were compared by one-way analysis of variance or t test, using SPSS Version 19.0 software (IBM Corporation, Armonk, NY). Values of $P < 0.05$ was considered to be significant.

Results

Cervical Lamina Block Therapy Promotes the Recovery of Clinical Vertigo and Improves Vertebrobasilar Artery Insufficiency

Vertigo symptoms in 34 out of 42 (81.0%) patients in the control group treated with vasodilator only were improved significantly, and 8 (19%) cases had no obvious improvements. In the experimental group in which patients were treated with vasodilator plus cervical lamina block therapy, there were 5 out of 48 (10%) cases with poor results, and the recovery rate was 89.6%. Although there was no significant statistical difference between the 2 groups in terms of DARS and VAS scores (Table 2), there was significant difference between the

2 groups in terms of hospital stays (P = 0.000), which indicates that cervical lamina block therapy benefits the recovery of clinical vertigo.

There was no significant difference comparing pretreatment VA flow in the control and experimental groups (P = 0.956). After treatment, the VA flow in the 2 groups were significantly different (P = 0.011 and P = 0.002 at 3 and 6 days after treatment). This indicates that cervical lamina block therapy could improve vertebrobasilar artery insufficiency, and this may explain the quick recovery of clinical vertigo.

According to the earlier mentioned clinical results, cervical lamina block therapy is an effective method to treat both neck pain and vertigo. Therefore, we think that the nerve innervation on cervical vertebra lamina could play an important part of connecting the neck and brain.

Electrical Stimulation of Cervical Ganglia Increases NE Expressions in Cervical Sympathetic Ganglia

We have previously reported a neural fiber connection between the spinal cervical and sympathetic ganglia. The earlier mentioned cervical lamina block therapy results indicated that a functional connection may exist between the 2 types of ganglia. As the adrenergic system is one of the important elements to regulate the blood flow of the vertebral arteries, we then tested if the adrenergic system was involved in the functional connection. NE, the main neurotransmitter of the adrenergic system to regulate the VA flow secreted by neuroendocrine cells in the sympathetic ganglia (6), is expressed in the superior cervical sympathetic ganglia (SCG) and inferior cervical sympathetic ganglia (ICG), and localized in neuronal nuclei as irregular brown granules. We analyzed if NE level in the sympathetic ganglia is affected by cervical spinal ganglia stimulation. NE level was assessed by immunohistochemistry and represented as MOD. After electrical stimulation of the cervical ganglia, the MOD values of NE in SCG of C2 to C3 groups increased significantly (F = 19.114 and F = 14.715; P = 0.000 and P = 0.001; Table 3) compared with that in the control group, and increased most when C2 spinal ganglia was stimulated. There was no significant difference among C2 to C3 groups (F = 1.603; P = 0.224) and among group C4 to C8 (F = 0.704; P = 0.594).

Significantly different with SCG, after electrical stimulation of the cervical spinal ganglia, the MOD values of NE in ICG of C6 to C8 groups increased significantly compared with that in the control group (F = 8.149, 21.294, and 28.364; P = 0.011, 0.000, and 0.000; Table 3). There was no significant difference among C6 to C8 groups (F = 0.770; P = 0.474) and between C2 and C5 groups (F = 0.791; P = 0.508).

Adrenergic Receptor Blockage Inhibits the Electrical Stimulation-Induced BA Flow Changes in Rabbits

As NE expression level in the cervical sympathetic ganglia was affected by the electrical stimulation of the cervical spinal ganglia, we then tested if NE mediated the functional connection between the 2 types of ganglia. Phentolamine, a adrenergic receptor blocker, was used to block adrenergic receptor around vertebral arteries. As shown in Table 4, electric stimulation to the spinal vertical ganglia accordingly decreased the BA flow (P = 0.000 in all groups). Adrenergic blocker treatment of VA significantly decreases the effect of electric stimulation on BA flow comparing with the saline solution treatment (P < 0.05 in all groups). These results indicated that NE is one important element of the cervical spinal ganglia regulating vertebrobasilar artery flow.

Table 2. *Data in clinical study.*

		Experimental Group n = 48	Control Group n = 42	*P* Values
VAS	pretherapy	6.792 ± 1.237	6.762 ± 1.620	0.922
	posttreatment	3.250 ± 1.139	3.262 ± 1.251	0.962
DARS	pretherapy	25.562 ± 6.441	26.405 ± 6.332	0.534
	posttreatment	12.354 ± 5.004	13.786 ± 5.550	0.202
Hospital stays (days)		7.250 ± 2.311	11.976 ± 2.561	0.000
Bilateral VA flow (mL/min)	pretherapy	165.854 ± 6.152	166.164 ± 6.604	0.818
	Third day after treatment	186.367 ± 3.977	180.364 ± 6.528	0.000
	Sixth day after treatment	193.865 ± 3.873	186.686 ± 5.462	0.000

Table 3. *NE expressions in control and normal experimental groups.*

Groups	n	NE Expression in SCG		NE Expression in ICG	
		MOD	*P* values	MOD	*P* values
C0	9	0.3643 ± 0.0388		0.3707 ± 0.0345	
C2	9	0.4525 ± 0.0465	0.000	0.3799 ± 0.0316	0.561
C3	9	0.4287 ± 0.0321	0.001	0.3658 ± 0.0390	0.782
C4	9	0.3916 ± 0.0572	0.253	0.3922 ± 0.0316	0.187
C5	9	0.3770 ± 0.0425	0.516	0.3865 ± 0.0485	0.437
C6	9	0.3739 ± 0.0347	0.586	0.4462 ± 0.0715	0.011
C7	9	0.3548 ± 0.0357	0.598	0.4569 ± 0.0442	0.000
C8	9	0.3738 ± 0.0370	0.601	0.4786 ± 0.0501	0.000

P values were the results of comparing normal experimental group with control group C0 using one-way analysis of variance.

Segmental Functional Connections Existed Between Cervical Spinal and Sympathetic Ganglia

As we found NE level in SCG or ICG was significantly affected by C2 to C3 or C6 to C8 spinal ganglia stimulation, segmental functional connections exist between the cervical spinal and sympathetic ganglia. In the second animal experiment, the poststimulation BA flow in C4 and C5 normal groups was significantly higher than that in other normal groups (F = 2.522; *P* = 0.031). These results indicated vertebrobasilar artery was more strongly regulated by C2 to C3 or C6 to C8 spinal ganglia via SCG or ICG.

Discussion

Cervical vertigo is characterized by vertigo from the cervical spine. However, whether cervical vertigo is an independent entity still remains controversial (7). Clinicians have no method to separate patients with vertigo caused by neck disorders from the patients who have both neck disorders and vertigo. The simultaneous existence of cervical vertigo and neck pain has been recognized by most experts (7-9), however, whether there is any relationship between the cervical vertigo and neck disorders remains controversial. We have reported bidirectional segmental nerve fiber connections between the cervical spinal and sympathetic ganglia (2). This manuscript further supports the existence of such connection between the cervical spinal and sympathetic ganglia by provided clinical and functional evidence, which might be used to explain the relationship between cervical vertigo and neck pain.

In the clinical study, we found that cervical lamina block therapy could significantly shorten the hospital stays. This therapy result was in agreement with the study conducted by Willburger et al (10) that cervical injection therapy could be effective with few side effects. The cervical lamina is mainly innervated by the spinal nerve, and facet joints are the most densely innervated among all the spinal joints (4). There are many mechanoreceptors in the deep segmental upper cervical muscles around the cervical lamina (3). Stimulation signals from neck tissues including direct trauma, degenerative changes, or neck pain can be accepted by the mechanoreceptors and sent to the central nervous system by the posterior ramus of the cervical nerve and spinal cord (11). The fact that cervical lamina block therapy could significantly relieve neck pain and vertigo and increase the BA flow indicated that signal transduction pathways could exist from the cervical nerve to the vertebrobasilar artery aside from the cervical nerve to sensorium. The posterior ramus of the cervical nerve are derived from the spinal ganglia connecting with the spinal cord. Therefore, the spinal ganglia could play an important role in this supposition pathway. As for the pathway of the spinal ganglia altering the vertebrobasilar artery, we performed the following animal experiments to verify it.

The sympathetic postganglionic fibers surrounding the vertebral arteries maintain arteries in a proper vasoconstriction degree under normal conditions. Pearce (12) reported that the sympathetic plexus could be stimulated by cervical degenerative diseases, and this stimulation could cause reflexive vasoconstriction of the vertebrobasilar system. NE is synthesized and secreted by neuroendocrine cells in the sympathetic ganglia, and serves as the most important neurotransmitter of the sympathetic postganglionic fibers (6). It regulates most arteries and veins to increase venous return and reduce

Table 4. *BA flow in animal experimental groups*.

Groups		n	Prestimulation (mL/min)	Poststimulation (mL/min)	*P* Values (pre vs. post)	
C2	Normal	9	25.0 ± 3.1‡	16.0 ± 2.6§	0.000	**P* = 0.013
	Saline treatment	9	23.8 ± 3.0*	15.4 ± 2.5†	0.000	†*P* = 0.003
	Phentolamine treatment	9	29.2 ± 4.8*	21.4 ± 4.5†	0.000	
C3	Normal	9	23.6 ± 2.5‡	15.5 ± 2.5§	0.000	**P* = 0.020
	Saline treatment	9	23.6 ± 3.4*	15.3 ± 2.8†	0.000	†*P* = 0.001
	Phentolamine treatment	9	28.2 ± 4.0*	21.1 ± 3.7†	0.000	
C4	Normal	9	23.6 ± 3.4‡	20.2 ± 3.2§	0.000	**P* = 0.033
	Saline treatment	9	24.3 ± 4.3*	19.1 ± 3.0†	0.000	†*P* = 0.025
	Phentolamine treatment	9	28.4 ± 2.8*	23.1 ± 3.8†	0.000	
C5	Normal	9	25.5 ± 4.4‡	19.1 ± 4.9§	0.000	**P* = 0.024
	Saline treatment	9	24.6 ± 4.2*	18.4 ± 4.4†	0.000	†*P* = 0.019
	Phentolamine treatment	9	29.0 ± 3.2*	22.6 ± 2.0†	0.000	
C6	Normal	9	25.3 ± 4.5‡	16.7 ± 3.1§	0.000	**P* = 0.044
	Saline treatment	9	23.8 ± 3.9*	14.5 ± 3.0†	0.000	†*P* = 0.014
	Phentolamine treatment	9	27.8 ± 4.0*	18.7 ± 3.4†	0.000	
C7	Normal	9	25.3 ± 5.1‡	15.6 ± 4.0§	0.000	**P* = 0.007
	Saline treatment	9	23.6 ± 3.0*	16.2 ± 2.5†	0.000	†*P* = 0.001
	Phentolamine treatment	9	29.0 ± 4.3*	20.3 ± 2.0†	0.000	
C8	Normal	9	25.2 ± 4.4‡	17.2 ± 3.0§	0.000	**P* = 0.022
	Saline treatment	9	24.3 ± 3.9*	16.4 ± 3.2†	0.000	†*P* = 0.029
	Phentolamine treatment	9	29.0 ± 4.0*	19.8 ± 2.8†	0.000	

‡$F = 0.380$, $P = 0.889$; §$F = 2.522$, $P = 0.031$, comparing all experimental groups.
*Phentolamine treatment of VA significantly increases the BA flow(all $P < 0.05$).
†Phentolamine treatment of VA significantly decreases the effect of electric stimulation on BA flow comparing with the aline treatment(all $P < 0.05$).

peripheral blood volume. The vertebrobasilar artery contraction then induces cerebral insufficiency and the symptom of vertigo. Thus, we chose NE expression level in the sympathetic ganglia to investigate the possible functional connection between the cervical spinal and sympathetic ganglia. In the animal experiment, the contents of NE in the ipsilateral SCG or ICG increased significantly when C2 to C3 or C6 to C8 spinal ganglia were electrically stimulated and the BA flow decreased. It was similar to the vertebrobasilar artery insufficiency in our clinical study, as Berger et al (13) reported that spinal cord trauma could cause sympathetic disorders and abnormal changes of blood pressure. After the adrenergic receptor blocker was injected surrounding VA, BA flow significantly increases comparing with the aline treatment. The fact that electrical stimulation to the cervical spinal ganglia could increase NE level in the cervical sympathetic ganglia and decrease BA flow further confirmed the possible connection pathway between the cervical spinal and sympathetic ganglia, and NE is one of the neurotransmitters of this pathway. It was proven that the adrenergic system could play a part of cervical spinal ganglia altering vertebrobasilar arteries.

Conclusions

Our report further confirmed the potential connection between the cervical spinal and sympathetic ganglia, and the adrenergic system may serve as a possible neurotransmitter. This could provide a neurochemical foundation for connection of neck pain and vertigo and a mechanism for the pathogenesis of cervical vertigo.

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