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Effect of Mecobalamin Combined with Edaravone on Nerve Conduction Velocity in Patients with Diabetic Peripheral Neuropathy

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Abstract: *This study aims to analyze the therapeutic effect of mecobalamin combined with edaravone in patients with diabetic peripheral neuropathy (DPN). A total of 65 patients with DPN treated in our hospital from May 2024 to May 2025 were randomly divided into a control group (32 cases, receiving conventional treatment plus mecobalamin) and an observation group (33 cases, receiving additional edaravone based on the control group regimen), and the treatment outcomes were compared between the two groups. The results showed that the observation group demonstrated significantly better nerve conduction velocity indices, serological markers, and vibration perception threshold (VPT) than the control group ($P < 0.05$). It is concluded that the combination of mecobalamin and edaravone can effectively improve nerve conduction velocity, serum indicators, and VPT in patients with DPN.*

Keywords: *Methylcobalamin; Edaravone; Diabetic peripheral neuropathy; Nerve conduction velocity; Vibration perception threshold*

1. Introduction

Diabetic peripheral neuropathy (DPN) is a common microangiopathy caused by the activation of polyol pathway, oxidative stress, lack of neurotrophic factors and other factors, which is characterized by Schwann cell injury, axonal degeneration and nerve blood supply disorders, and ultimately by nerve conduction dysfunction[1]. The main manifestations of DPN patients are symmetrical distal paresthesia (numbness, tingling, burning sensation, etc.), sensory loss, foot ulcers and gangrene, which reduce the quality of life, and significantly increase the burden of family and society[2]. At present, the clinical treatment of DPN is based on the control of blood glucose and the intervention of nerve injury drugs, in which mecobalamin is commonly used, which can repair damaged nerves by promoting myelination and axonal regeneration, but it is difficult to inhibit oxidative stress and microcirculation disorders caused by DPN, and can not improve nerve conduction velocity[3]. Edaravone is a powerful free radical scavenger, which can inhibit lipid peroxidation and reduce oxidative damage of vascular endothelial cells and neurons[4]. The combination of the two can play a synergistic role in repairing nerves and resisting oxidative stress, thus improving the nervous microenvironment in an all-round way. This study analyzed the effect of mecobalamin combined with edaravone in patients with DPN, aiming to evaluate the clinical effect of the combination regimen and provide evidence-based evidence for optimizing the drug treatment strategy of DPN. Elaborated as follows.

2. Data and Methods

2.1 General information

Sixty-five patients with DPN treated in our hospital from May 2024 to May 2025 were randomly selected and divided into double-blind groups. The control group (n = 32) was 40-78 years old, mean (62.15 ± 5.38) years old, male/female (20/12); the observation group (n = 33) was 35-80 years old, mean (61.84 ± 5.42) years old, male/female (18/15). Comparison of general data showed no statistically significant differences between the two groups ($P > 0.05$).

Inclusion criteria: age > 18 years old, diagnosed as DPN, no other drug treatment for DPN except hypoglycemic drugs one month before enrollment, complete data, voluntary signing of informed consent, good compliance.

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Exclusion criteria: patients with peripheral neuropathy caused by other reasons, patients with severe heart, liver and kidney dysfunction, patients with malignant tumors, patients with diabetic ketoacidosis, pregnant women, and patients allergic to the drugs in this study.

2.2 Method

2.2.1 Control group

The control group was treated with conventional therapy and mecobalamin. According to the results of blood glucose monitoring, patients were given hypoglycemic drugs or insulin therapy to ensure FBG ≤ 7.0mmoL/L \ 2 hPBG ≤ 10.0mmoL/L. Intravenous injection of methylcobalamin 500 μg, once a day. The treatment lasted for 10 days.

2.2.2 Observation group

The observation group was treated with edaravone combined with the control group. Intravenous infusion of 30 mg + 100 ml saline, twice a day. The treatment lasted for 10 days.

2.3 Index observation

(1) Nerve conduction velocity: Motor nerve conduction velocity (MNCV) and sensory nerve conduction velocity (SNCV) of median nerve and common peroneal nerve were detected by electromyography evoked potential instrument before treatment and 10 days after treatment. (2) Serological indexes: 3ml venous blood was centrifuged (speed 3000r/min, time 10 min), SOD, GSH-Px and MDA were detected by enzyme-linked immunosorbent assay before and 10 days after treatment, and HCY was detected by radioimmunoassay before and 10 days after treatment. (3) VPT: VPT (vibration perception threshold) was used to monitor the VPT of the left and right limbs before treatment and at rest for 10 days.

2.4 Statistical analysis

The data were processed by SPSS 26.0 software, and the enumeration data were expressed as [n (%)], and the 2 test was adopted. The measurement data shall be ($\overline{x} \pm s$), and t-test is adopted. $P < 0.05$ indicates a statistically significant difference.

3. Results

3.1 Comparison of nerve conduction velocity index

The nerve conduction velocity indexes of the two groups before treatment were compared ($P > 0.05$), and the observation group was higher than control group after 10 days of treatment ($P < 0.05$), as shown in Table 1.

Table 1. Comparison of nerve conduction velocity indexes [$\overline{x} \pm s$ (m/s)]

Grouping	Number of cases	Median nerve MNCV		Median nerve SNCV		Common peroneal nerve MNCV		Common peroneal nerve SNCV	
		Before treatment	The treatment lasted for 10 days	Before treatment	The treatment lasted for 10 days	Before treatment	The treatment lasted for 10 days	Before treatment	The treatment lasted for 10 days
Observation group	33	41.68±4.12	55.59±5.34*	35.51±3.23	45.76±4.05*	40.26±4.47	49.24±4.82*	35.35±3.42	45.93±4.26*
Control group	32	41.96±4.23	48.25±5.08*	35.89±3.45	40.13±4.01*	40.53±4.61	45.07±4.50*	35.03±3.46	40.32±4.08*
T-value	-	0.270	5.674	0.458	5.630	0.239	3.602	0.374	5.419
P value	-	0.787	0.000	0.648	0.000	0.811	0.000	0.708	0.000

Note: Compared with this group before treatment*, $P < 0.05$.

3.2 Comparison of serological indicators

The serological indexes of the two groups before treatment were compared ($P > 0.05$), and the observation group was better than control group after 10 days of treatment ($P < 0.05$), as shown in Table 2.

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Table 2. Comparison of serological indices [$\bar{x} \pm s$]

Grouping	Number of cases	SOD(KU/L)		GSH-Px(KU/L)		MDA(nmol/L)		HCY(mmol/L)	
		Before treatment	The treatment lasted for 10 days	Before treatment	The treatment lasted for 10 days	Before treatment	The treatment lasted for 10 days	Before treatment	The treatment lasted for 10 days
Observation group	33	70.34±7.16	82.41±8.05*	6.61±0.75	4.16±0.61*	112.24±10.54	127.96±12.13*	25.23±2.47	12.79±1.64*
Control group	32	70.98±7.23	75.16±7.32*	6.52±0.74	5.23±0.78*	110.18±10.25	120.32±11.78*	25.06±2.28	17.65±1.96*
T-value	-	0.358	3.795	0.486	6.171	0.798	2.574	0.288	10.855
P value	-	0.721	0.000	0.628	0.000	0.427	0.012	0.774	0.000

Note: Compared with this group before treatment*, P < 0.05. Superoxide dismutase (SOD), glutathione peroxidase (GSH-Px), malondialdehyde (MDA), and homocysteine (HCY) were measured by radioimmunoassay.

3.3 Comparison VPT

Before treatment, the VPT of the left and right limbs of the two groups was compared (P > 0.05), and the observation group was lower than control group at 10 days after treatment (P < 0.05), as shown in Table 3.

Table 3. vs. VPT [$\bar{x} \pm s$ (V)]

Grouping	Number of cases	Left lower extremity		Right lower extremity	
		Before treatment	The treatment lasted for 10 days	Before treatment	The treatment lasted for 10 days
Observation group	33	20.12±3.34	16.48±2.51*	20.45±3.12	16.14±1.82*
Control group	32	20.34±3.28	18.59±2.87*	20.34±3.17	18.95±1.98*
T-value	-	0.267	3.157	0.140	5.959
P value	-	0.789	0.002	0.888	0.000

Note: Compared with this group before treatment*, P < 0.05.

4. Conclusion

The results of this study showed that the nerve conduction velocity of the observation group was higher than that of the control group after 10 days of treatment (P < 0.05), which confirmed that the nerve conduction velocity of DPN patients could be improved by the combination of mecobalamin and edaravone. Mecobalamin combined with edaravone plays a synergistic role in precise intervention of different pathological links of DPN. Mecobalamin can directly penetrate the blood-nerve barrier into nerve cells, participate in one-carbon unit circulation and methylation reaction, promote the synthesis of nucleic acid and protein, thus promoting the reconstruction of damaged axon structure and repair of myelin sheath, and lay a material basis for the improvement of nerve conduction velocity[5]. As a powerful scavenger of hydroxyl free radicals, edaravone can precisely intervene in oxidative stress and improve hyperglycemia, thereby reducing the number of oxygen free radicals, inhibiting the accumulation of malondialdehyde, reducing the activity of superoxide dismutase and the number of apoptosis, improving the oxidative damage of nerve tissue and the function of nerve capillaries. Finally, it protects nerve axons from the continuous invasion of oxidative stress[6]. Edaravone can reduce the oxidative damage of vascular endothelial cells, improve the blood supply of endoneurium, and create a favorable microenvironment for nerve repair[7]. The combination of the two is not a simple superposition of pharmacological effects, but a dual protective network of structural repair and oxidative intervention: mecobalamin promotes the reconstruction of damaged nerve structure and repair of myelin sheath, while edaravone inhibits the oxidative stress waterfall reaction leading to nerve injury from the source and improves nerve blood supply. The two drugs play a sequential synergistic effect on different pathological links of DPN, so that the conduction velocity of motor and sensory nerves of median nerve and common peroneal nerve can be comprehensively improved.

After 10 days of treatment, the serological indexes of the observation group were better than those of the control group (P < 0.05), indicating that the improvement of serological indexes in patients with DPN combined with mecobalamin and edaravone. Mecobalamin combined with edaravone plays a synergistic regulatory mechanism at the molecular level, and they intervene from the multi-dimensional perspective of enhancing antioxidant enzyme activity, improving oxidative damage and remodeling

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methylation metabolism. Oxidative stress induced by hyperglycemia is one of the main inducements of DPN, which is characterized by the generation of a large number of oxygen free radicals, which are difficult to be effectively eliminated by the body itself, specifically reflected in the abnormal levels of serological indicators. Edaravone can directly penetrate the cell membrane, precisely capture and scavenge oxygen free radicals, thus inhibiting the attack of free radical chain reaction on the lipid membrane, and ultimately reducing MDA levels[8]. Edaravone has antioxidant effect, which can inhibit the damage of oxidative stress to enzyme protein, increase the levels of SOD and GSH-Px, and thus maintain the functional reserve of endogenous antioxidant system[9]. Methylcobalamin participates in the remethylation metabolism of homocysteine, promotes its conversion to methionine, reduces the level of HCY, fundamentally improves the secondary oxidative stress triggered by hyperhomocysteinemia, and forms a virtuous circle of "reducing HCY-reducing oxidative burden". Methylcobalamin promotes the synthesis of nucleic acid and protein, provides sufficient substrate support for the synthesis of SOD and GSH-Px of antioxidant enzymes, and promotes the reconstruction of antioxidant system from the anabolic level. The combination of the two shows the synergistic effect of "edaravone cures the symptoms and mecobalamin cures the root cause": edaravone scavenges oxygen free radicals and protects the existing enzyme activity, and rapidly reduces the level of MDA; mecobalamin reduces the generation of free radicals by improving the metabolism of homocysteine, provides raw material support for the synthesis of antioxidant enzymes, and jointly achieves the synergistic recovery of SOD and GSH-Px levels.

The VPT of left and right limbs in the observation group was lower than that in the control group after 10 days of treatment ($P < 0.05$), which proved that the combination of mecobalamin and edaravone could reduce the vibration sensation in patients with DPN. Mecobalamin combined with edaravone has synergistic protective effects on the structure and function of large-diameter nerve fibers. VPT is a core index for clinical evaluation of the integrity of the deep sensory conduction pathway, which mainly reflects the functional status of Aβ thick myelinated nerve fibers and the sensory efficacy of the Pacinian bodies connected to them. The increase of VPT indicates the pathological changes of demyelination, axonal degeneration or both of the myelinated nerve fibers. Methylcobalamin is a coenzyme of methionine synthetase, which participates in the repair of damaged myelin structure by promoting the synthesis of myelin basic protein and myelin lipid, regulates the transport of axonal skeleton protein, and stabilizes the axonal microtubule structure, thus providing a structural basis for the transmission of vibration signals. Edaravone can reduce the lipid peroxidation damage of Schwann cells and axon membrane and protect the activity of Na + -K + -ATPase by scavenging hydroxyl free radicals, thus maintaining the normal generation and distribution of resting potential and action potential of nerve fibers[10]. Edaravone can protect nerve microvascular endothelial cells, improve endoneurial blood perfusion and nerve ischemia and hypoxia. In combination, mecobalamin provides raw materials for repairing myelin sheath and axon at the molecular level, while edaravone escorts the repair process by improving the nerve microenvironment and directly protects the excitability and conductivity of nerve fibers. Through the dual intervention of microstructure remodeling and overall function maintenance, the mechanical stimulation threshold of thick myelinated fibers gradually fell back to the normal range, and finally the VPT values of left and right limbs decreased.

To sum up, mecobalamin combined with edaravone is helpful to improve nerve conduction velocity, serological indexes and VPT in patients with DPN.

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