

Clinical Effect of Memantine Hydrochloride Combined with Carbidopa-levodopa Sustained Release Tablet on Motor Symptoms in Patients with Parkinson's Disease

Jiayi Gu ^{1,#}, Xiuqing Fu ^{1,#}, Qiaowen Tong ¹, Yiting Ni ¹, Hua Ye ^{1,*}

¹. Department of Neurology, Wenzhou People's Hospital, 325000 Wenzhou, Zhejiang, China

These authors contributed equally to this work.

DOI: <https://doi.org/10.62767/dbm601.7464>

Keywords

Memantine hydrochloride
Carbidopa-levodopa sustained release tablet
Parkinson's disease
Motor symptoms

* Correspondence

Hua Ye
Department of Neurology, Wenzhou People's Hospital, 325000 Wenzhou, Zhejiang, China
E-mail: yehua0577@163.com

Received: 25 March 2026

Revised: 9 May 2026

Accepted: 12 May 2026

Published: 17 July 2026

Diagnostic Brain Medicine 2026; 6(1): 1-10.

Abstract

Background: To investigate the clinical effect of memantine hydrochloride combined with carbidopa-levodopa sustained release tablet on motor symptoms in patients with Parkinson's disease. **Methods:** 45 patients with Parkinson's disease receiving treatment of memantine hydrochloride combined with carbidopa-levodopa sustained release tablet in our hospital from September 25th, 2025 to December 25th, 2025 were included as the study group, and 45 Parkinson's disease patients treated with carbidopa-levodopa sustained release tablet were included as the control group during the same period. The clinical efficacy, adverse reactions, and changes of motor symptoms and serum indexes before and after treatment were compared between the two groups. **Results:** The total effective rate of clinical efficacy in the study group was higher than that in the control group ($P < 0.05$). After treatment, UPDRS-III scores and levels of IL-1 β , IL-17, Hcy, TNF- α and MDA were decreased in both groups, and were lower in the study group than the control group ($P < 0.05$). The levels of SOD and GSH-Px in both groups were increased, and were higher in the study group than the control group ($P < 0.05$). There was no significant difference in gastrointestinal reaction, lethargy, dizziness, headache and postural hypotension between the two groups ($P > 0.05$). **Conclusion:** Memantine hydrochloride combined with carbidopa-levodopa sustained release tablets has good clinical effects in the treatment of Parkinson's disease through reducing motor symptoms, inhibiting inflammatory response, improving oxidative stress, etc., with good safety.



1 Introduction

Parkinson's disease is a neurodegenerative disorder with the middle-aged and elderly individuals as the main sufferers, which is caused by the degeneration and death of dopaminergic neurons in the substantia nigra, leading to a deficiency in dopamine synthesis. Patients often exhibit motor symptoms such as resting tremor, muscle rigidity, and unstable gait, as well as non-motor symptoms like cognitive impairment and sleep disturbances [1,2].

Currently, there is no standard treatment regimen for Parkinson's disease in clinical practice. The main treatment strategies include pharmacological therapy, surgical intervention, and rehabilitation training. Pharmacological therapy remains the cornerstone of Parkinson's disease management, primarily comprising levodopa preparations, dopamine receptor agonists, monoamine oxidase B inhibitors, catechol-O-methyltransferase inhibitors, and anticholinergic drugs [3]. Levodopa combined with carbidopa is considered the gold standard for symptomatic treatment, effectively improving motor symptoms [4]. However, long-term levodopa therapy is associated with motor complications such as wearing-off phenomena and dyskinesia [5]. Dopamine agonists, including pramipexole and ropinirole, are commonly used as initial monotherapy or adjunctive therapy to delay levodopa-related complications [6]. Additionally, deep brain stimulation surgery has emerged as an effective treatment option for advanced Parkinson's disease patients with severe motor fluctuations or dyskinesia [7].

Given the limitations of monotherapy, combining different medications has become an important strategy to optimize therapeutic outcomes and reduce adverse effects. Carbidopa-levodopa sustained release tablets, also known as Xining, are the main drugs for dopamine replacement therapy, which can be converted into dopamine under the action of enzymes.

By increasing the concentration of dopamine in the patient's brain and inhibiting the overactivity of cholinergic neurons, the tablets decelerate the progression of Parkinson's disease [8]. However, studies have shown that the efficacy of carbidopa-levodopa sustained release tablets may gradually decrease with long-term use, and may also result in adverse reactions such as nausea, vomiting, and cardiovascular events [9]. Therefore, combining other drug for treating Parkinson's disease to improve clinical outcomes and reduce the risk of adverse reactions has been clinically considered. Memantine hydrochloride is a new type of non-competitive receptor antagonist that can exert neuroprotective effects by antagonizing the toxicity of excitatory amino acids [10]. Memantine hydrochloride can significantly improve the clinical symptoms of Parkinson's disease patients with good therapeutic effects [11]. Based on this, this study applies memantine hydrochloride combined with carbidopa-levodopa sustained release tablets for the treatment of Parkinson's disease patients, and determines their application value by observing the clinical effects and comparing their impacts on motor symptoms, inflammation, oxidative stress responses, etc., thereby providing more references and evidence for the clinical treatment of Parkinson's disease.

2 Materials and methods

2.1 General information

45 patients with Parkinson's disease receiving treatment of memantine hydrochloride combined with carbidopa-levodopa sustained release tablet in our hospital from September 25th, 2025 to December 25th, 2025 were included as the study group, and 45 Parkinson's disease patients treated with carbidopa-levodopa sustained release tablet were included as the control group during the same period. There was no statistically significant difference in gender, age, disease duration, clinical symptom

classification, Hoehn-Yahr grading, and comorbidities between the two groups of patients ($P > 0.05$), which were comparable (Table 1). This study was approved

by the Ethics Committee of Wenzhou People's Hospital (Ethics Approval No. KY-202509-180). All patients signed informed consent forms before enrollment.

Table 1 Comparison of general information between the two groups.

Project	Study group (n=45)	Control group (n=45)	$t/Z/\chi^2$	P
Gender [case (%)]			0.179	0.673
Male	23 (51.11)	25 (55.56)		
Female	22 (48.89)	20 (44.44)		
Age (year)	64.71±4.55	65.58±5.90	-0.781	0.437
Disease course (month)	60.27±11.96	62.42±12.70	-0.829	0.409
Clinical symptom classification [case (%)]			0.530	0.767
Rigidity with little movement type	19 (42.22)	18 (40.00)		
Tremor type	15 (33.33)	13 (28.89)		
Mixed type	11 (24.44)	14 (31.11)		
Hoehn-Yahr grading [case (%)]			-0.088	0.930
Grade II	12 (26.67)	16 (35.56)		
Grade III	26 (57.78)	18 (40.00)		
Grade IV	7 (15.56)	11 (24.44)		
Comorbidities [case (%)]				
Hypertention	18 (40.00)	16 (35.56)	0.189	0.664
Diabetes	10 (22.22)	12 (26.67)	0.241	0.624
Coronary heart disease	9 (20.00)	11 (24.44)	0.257	0.612

2.2 Inclusion and exclusion criteria

2.2.1 Inclusion criteria

(1) Meeting the diagnostic criteria for Parkinson's disease as outlined in the "Chinese Diagnostic Criteria for Parkinson's Disease (2016 edition)" [12]; (2) not participating in any other clinical or academic studies during the treatment period.

2.2.2 Exclusion criteria

(1) Symptomatic Parkinson's disease or Parkinson's disease with syndromes; (2) Severe peak-dose dyskinesia; (3) A history of cranial organic injury or other central nervous system diseases; (4) Insufficiency of important organs such as the heart, lungs, liver, or kidneys; (5) Mental disorders or

dementia; (6) A history of drug abuse or alcoholism; (7) Allergy to the medications used in this study.

2.3 Methods

2.3.1 Control group

Patients in the control group orally took carbidopa-levodopa sustained release tablets (MSD, Hangzhou, China; National medicine permission number (NMPN): HJ20160371; 250 mg/tablet), 125 mg/time and 2 times/day for 12 weeks.

2.3.2 Study group

Based on the treatment in the control group, patients in the study group additionally orally took memantine hydrochloride (CSPC, Hebei, China; NMPN: H20203320; 5 mg) at 5, 10, 15 and 20 mg/d in the

first, second, third and fourth weeks, respectively, and then maintained at 20 mg/d from the 5th week to the 12th week.

2.4 Clinical indicators

2.4.1 Clinical efficacy

Clinical outcomes were assessed using the Unified Parkinson's Disease Rating Scale (UPDRS-III). Criteria: Cured: complete resolution of symptoms, with an improvement rate in UPDRS-III of $\geq 95\%$; Markedly effective: significant improvement in symptoms, with an improvement rate in UPDRS-III of 70% to 94%; Improved: some improvement in symptoms, with an improvement rate in UPDRS-III of 20% to 69%; Ineffective: not meeting the above criteria. The total clinical efficacy rate = (number of cured + markedly effective + improved cases)/total number of cases $\times 100\%$.

2.4.2 Motor symptoms

Motor symptoms were evaluated using the UPDRS-III before treatment and after 12 weeks of treatment. The scale uses a 5-point rating system from 0 to 4, including 14 items such as speech, facial expression, and resting tremor, with higher scores indicating more severe motor symptoms [13].

2.4.3 Serum biomarkers

Before treatment and after 12 weeks of treatment, 5 ml fasting peripheral venous blood was collected from both groups of patients in the early morning, left to stand at room temperature for 30 to 60 min, and centrifuged at 3000r/min for 10 min. The separated serum was stored at -20°C for later testing. Enzyme-linked immunosorbent assay (ELISA) was used with reagent kits purchased from Beijing Bio-Lab Technology Co., Ltd. to measure the levels of interleukin (IL)-1 β , IL-17, homocysteine (Hcy), tumor necrosis factor (TNF)- α , and malondialdehyde (MDA). Visible spectrophotometry and ultraviolet

spectrophotometry were applied with reagent kits purchased from Shanghai Jizhi Biochemical Technology Co., Ltd. to measure the levels of superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px), respectively. All operations were strictly performed according to the instructions.

2.4.4 Adverse reactions

The occurrence of gastrointestinal reactions, lethargy, dizziness, headache, and postural hypotension during the treatment period in both groups of patients was statistically recorded and documented.

2.5 Sample size calculation

The sample size was calculated based on the improvement in the primary outcome measure, the UPDRS-III score. Referring to the randomized controlled trial design conducted by Han L et al. [14] on the treatment of motor dysfunction in Parkinson's disease with Jiaji Panlong needling, the post-treatment UPDRS-III scores of the two groups were (25.34 ± 4.72) points and (28.67 ± 5.08) points, respectively. The PASS 2021 software was adopted for sample size calculation, with the statistical power set as $1-\beta = 0.90$ and the significance level $\alpha = 0.05$. The calculation results indicated that a minimum of 21 patients were required in each group. Considering a 20% dropout rate, the final sample size was determined to be 27 patients per group, with a total of no fewer than 54 patients enrolled in the two groups.

2.6 Statistical methods

Statistical analysis was performed using SPSS 26.0. Measurement data underwent normality analyses using the Kolmogorov-Smirnov test. Normally distributed continuous variables were expressed as mean $\pm$ standard deviation ($\bar{x} \pm s$), and comparisons between groups were made using independent samples t-tests, while comparisons within groups at two time points were completed using paired samples t-tests. Non-normally distributed continuous variables

were expressed by quartile method [M (P25, P75)], and comparisons between groups were achieved using the Mann-Whitney U test, while comparisons within groups at two time points were made using the Wilcoxon signed-rank test. Categorical data were displayed as rates or proportions and were compared using the χ^2 test. Comparisons between two groups for ordinal data were made using the Mann-Whitney U

test. A P-value of less than 0.05 was considered statistically significant.

3 Results

3.1 Comparison of clinical efficacy between the two groups

The total clinical effective rate was higher in the study group than the control group ($P < 0.05$, Table 2).

Table 2 Comparison of clinical efficacy between the two groups [case (%)].

Group	Case	Cured	Markedly improved	Improved	Ineffective	Total effective rate
Study group	45	2 (4.44)	12 (26.67)	22 (48.89)	9 (20.00)	36 (80.00)
Control group	45	0 (0.00)	4 (8.89)	22 (48.89)	19 (42.22)	26 (57.78)
Z				-2.972		
P				0.003		

3.2 Comparison of motor symptoms before and after treatment between the two groups

Before treatment, there was no statistically significant difference in UPDRS-III scores between the two

groups of patients ($P > 0.05$). After treatment, the UPDRS-III scores in both groups were decreased ($P < 0.05$), and were lower in the study group than the control group ($P < 0.05$, Table 3).

Table 3 Comparison of UPDRS-III before and after treatment between the two groups (score).

Group	Case	Before treatment	After treatment
Study group	45	37.47±13.02	17.00 (12.00, 20.00)*
Control group	45	36.44±13.94	25.00 (16.00, 31.00)*
t/Z		0.360	-3.299
P		0.720	<0.001

Compared to before treatment, * $P < 0.05$

3.3 Comparison of serum indicators before and after treatment between the two groups

Without treatment, the levels of IL-1 β , IL-17, Hcy, TNF- α , MDA, SOD, and GSH Px between the two groups displayed no significant difference ($P > 0.05$).

Following treatment, the levels of IL-1 β , IL-17, Hcy, TNF- α , and MDA were diminished, while levels of SOD and GSH Px were elevated in both groups ($P < 0.05$), and the changes were more evident in the study group than the control group ($P < 0.05$), as shown in Table 4.

Table 4 Comparison of serum indicators before and after treatment between the two groups ($\bar{x} \pm s$).

Group	Case	IL-1 β (ng/mL)		IL-17 (pg/mL)	
		Before treatment	After treatment	Before treatment	After treatment
Study group	45	0.63±0.15	0.32±0.17*	17.08±4.99	8.50±2.35*
Control group	45	0.64±0.17	0.47±0.13*	18.69±4.46	12.32±2.98*
t		-0.138	-4.846	-1.620	-6.745

<i>P</i>	0.890	<0.001	0.108	<0.001
----------	-------	--------	-------	--------

Compared to before treatment, **P* < 0.05

(Continued) **Table 4** Comparison of serum indicators before and after treatment between the two groups ($\bar{x} \pm s$).

Group	Case	Hcy ($\mu\text{mol/L}$)		TNF- α (ng/L)	
		Before treatment	After treatment	Before treatment	After treatment
Study group	45	28.13 $\pm$ 7.20	11.51 $\pm$ 3.36*	2.62 $\pm$ 0.76	1.60 $\pm$ 0.34*
Control group	45	25.61 $\pm$ 5.94	17.83 $\pm$ 3.90*	2.55 $\pm$ 0.64	2.25 $\pm$ 0.38*
<i>t</i>		1.813	-8.228	0.455	-8.538
<i>P</i>		0.073	<0.001	0.650	<0.001

Compared to before treatment, **P* < 0.05

(Continued) **Table 4** Comparison of serum indicators before and after treatment between the two groups ($\bar{x} \pm s$).

Group	Case	SOD (U/L)		MDA (nmol/mL)		GSH-Px (U/L)	
		Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Study group	45	103.30 $\pm$ 9.89	139.07 $\pm$ 13.01*	7.26 $\pm$ 1.57	3.16 $\pm$ 0.74*	26.75 $\pm$ 7.52	41.25 $\pm$ 11.82*
Control group	45	102.69 $\pm$ 9.16	122.65 $\pm$ 11.99*	6.68 $\pm$ 1.62	5.58 $\pm$ 0.71*	24.56 $\pm$ 8.87	33.35 $\pm$ 7.64*
<i>t</i>		0.303	6.224	1.752	-15.851	1.265	3.769
<i>P</i>		0.762	<0.001	0.083	<0.001	0.209	<0.001

Compared to before treatment, **P* < 0.05

3.4 Comparison of adverse reactions between the two groups

There was no statistically significant difference in the

occurrence of gastrointestinal reactions, lethargy, dizziness and headache, and postural hypotension between the two groups of patients (*P* > 0.05, [Table 5](#)).

Table 5 Comparison of adverse reactions between the two groups [case (%)].

Group	Case	Gastrointestinal reactions	Lethargy	Dizziness and headache	Postural hypotension	Total occurrence rate
Study group	45	2 (4.44)	2 (4.44)	3 (6.67)	1 (2.22)	8 (17.78)
Control group	45	2 (4.44)	3 (6.67)	3 (6.67)	2 (4.44)	10 (22.22)
χ^2						0.278
<i>P</i>						0.598

4 Discussion

To investigate the clinical effects of memantine

hydrochloride combined with carbidopa-levodopa sustained release tablet in the treatment of Parkinson'

s disease patients, this study applied a combination therapy of memantine hydrochloride and carbidopa-levodopa sustained release tablet, as well as monotherapy with carbidopa-levodopa sustained release tablet, to two groups of patients. The results revealed that the combination therapy had superior clinical outcomes compared to monotherapy.

Motor symptoms are common clinical manifestations of Parkinson's disease, primarily including tremors, muscle rigidity, and bradykinesia, which are caused by dopamine synthesis reduction-induced physiological dysfunction of the nervous system and enhanced excitatory effects of acetylcholine; therefore, the severity of motor symptoms is closely related to the progression of the disease [15]. The findings of this study indicated that the combination therapy of memantine hydrochloride and carbidopa-levodopa sustained release tablet can alleviate motor symptoms in Parkinson's disease patients, with better effects compared to monotherapy with carbidopa-levodopa sustained release tablets. Memantine hydrochloride can antagonize N-methyl-D-aspartate (NMDA) receptors, reduce receptor excitability, and thereby decrease neuronal apoptosis, which can also augment the concentration of brain-derived neurotrophic factor in the cerebral cortex and exert neuroprotective effects, thus delaying the progression of Parkinson's disease and effectively improving motor symptoms [16]. Additionally, memantine hydrochloride can promote the release of dopamine and directly/indirectly excite dopamine receptors, thereby improving tremors and bradykinesia [17]. Memantine hydrochloride can significantly relieve motor symptoms in Parkinson's disease patients [18], consistent with the results of this study. Accordingly, the combination therapy of memantine hydrochloride and carbidopa-levodopa sustained release tablet was effective in alleviating motor symptoms in Parkinson's disease patients.

Studies have proved that overactivation of microglia plays a significant role in the progression of Parkinson's disease, leading to neuroinflammatory responses and oxidative stress [19,20]. The combination therapy exerts synergistic anti-inflammatory effects through complementary mechanisms: carbidopa-levodopa indirectly reduces neuroinflammation by restoring dopaminergic neurotransmission and inhibiting microglial activation, while memantine hydrochloride directly blocks NMDA receptor-mediated microglial activation and subsequent NF- κ B signaling, thereby suppressing pro-inflammatory cytokine production (IL-1 β , IL-17, TNF- α). Inflammatory factors such as IL-1 β can boost microglial activity and induce nitric oxide production, thereby damaging dopaminergic neuronal cells. They can also directly exert cytotoxic effects, causing neuronal damage, neuronal apoptosis and neurodegeneration, and exacerbating the condition of Parkinson's disease [21,22]. Oxidative stress is instrumental in the process of neuronal degeneration in Parkinson's disease, characterized by an imbalance between oxidative and antioxidant systems, resulting in the accumulation of reactive oxygen species and cytotoxicity, and contributing to the occurrence of neurodegenerative changes [23-25]. Hence, this study suggested that clinical treatment of Parkinson's disease can focus on inhibiting inflammatory responses and improving oxidative stress. Herein, we demonstrated that the combination therapy of memantine hydrochloride and carbidopa-levodopa sustained release tablets can dampen inflammatory responses and improve oxidative stress in Parkinson's disease patients, which was superior to monotherapy with carbidopa-levodopa sustained release tablets in terms of clinical effects.

Memantine hydrochloride has the ability to suppress inflammatory responses in Parkinson's disease patients [18] and can increase the levels of SOD and GSH-Px while decreasing the levels of MDA [26],

consistent with the results of this study. Notably, a recent experimental study by Choi et al. demonstrated that memantine attenuates TNF- α and IL-6 secretion through downregulation of TLR4 and I κ B signaling in microglial cells and improves motor coordination in a synucleinopathy mouse model, providing mechanistic support for our clinical observations [27]. While their study focused on the neuroinflammatory mechanisms in preclinical models, our clinical trial extends these findings to human patients, showing significant reductions in IL-1 β , IL-17, and TNF- α levels alongside motor symptom improvement. Furthermore, previous clinical trials of memantine in Parkinson's disease have primarily investigated monotherapy for axial symptoms or dementia, whereas our study demonstrates the enhanced efficacy of memantine combined with carbidopa-levodopa sustained release tablets on comprehensive motor symptoms, representing a novel therapeutic strategy with both symptomatic and neuroprotective benefits. It was evident that the combination therapy of memantine hydrochloride and carbidopa-levodopa sustained release tablets can effectively repress inflammatory responses and improve oxidative stress in Parkinson's disease patients.

Moreover, treatment of Parkinson's disease with carbidopa-levodopa sustained release tablets may trigger adverse reactions such as gastrointestinal reactions and dizziness [28]. Our results revealed no significant increase in the incidence of adverse reactions after combination therapy of memantine hydrochloride and carbidopa-levodopa sustained release tablets compared to monotherapy of carbidopa-levodopa sustained release tablets, implying that the combined therapy had a certain level of safety and did not raise the risk of reaction occurrence.

However, this study has several limitations. First, the sample size was relatively small ($n=90$), which may

limit the generalizability of the findings. Second, this was a single-center study, potentially introducing selection bias and limiting the representativeness of the patient population. Third, the observation period was relatively short (12 weeks), which may not be sufficient to fully evaluate the long-term efficacy and safety of the combination therapy, particularly regarding the delayed onset of adverse effects or the development of motor complications such as dyskinesia. Additionally, the lack of a placebo control group and the non-randomized design may affect the strength of the evidence. Future studies should expand the sample size and extend the observation period to further investigate the safety of the combination therapy of memantine hydrochloride and carbidopa-levodopa sustained release tablets in Parkinson's disease.

5 Conclusions

In summary, the combination therapy of memantine hydrochloride and carbidopa-levodopa sustained release tablets for Parkinson's disease demonstrates good clinical efficacy and plays a role in alleviating motor symptoms, inhibiting inflammatory responses, and improving oxidative stress, with a favorable safety profile.

Acknowledgements

Not applicable.

Conflicts of Interest

The authors declare no conflict of interest.

Author Contributions

J.G. and X.F. conceived and designed the study, conducted experiments, and analyzed data; Q.T. collected and verified research data; Y.N. supplied experimental resources and obtained funding; H.Y. supervised the whole research, managed the project and secured funding. All authors drafted and critically revised the manuscript, and approved the final version

for publication.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of Wenzhou People's Hospital (Ethics Approval No. KY-202509-180)

Funding

Not applicable.

Availability of Data and Materials

The analyzed data sets generated during the study are available from the corresponding author on reasonable request.

Supplementary Materials

Not applicable.

References

- [1] Huang Y, Chen X. Efficacy and potential mechanisms of Bushen Huoxue Shugan Decoction combined with Western medicine on Parkinson's disease. *Hebei Journal of Traditional Chinese Medicine* 2024; 46(4): 574-577+582.
- [2] Magrinelli F, Picelli A, Tocco P, et al. Pathophysiology of Motor Dysfunction in Parkinson's Disease as the Rationale for Drug Treatment and Rehabilitation. *Parkinson's Disease* 2016; 6(6): 9832839.
- [3] Kalia LV, Lang AE. Parkinson's disease. *The Lancet* 2015; 386(9996): 896-912.
- [4] Poewe W, Seppi K, Tanner CM, et al. Parkinson disease. *Nature Reviews Disease Primers* 2017, 23(3): 17013.
- [5] Morgan J, Sethi KD. Levodopa and the progression of Parkinson's disease. *Current Neurology and Neuroscience Reports* 2005; 5(4): 261-2.
- [6] Schapira AHV, Chaudhuri KR, Jenner P. Non-motor features of Parkinson disease. *Nature Reviews Neuroscience* 2017, 18(7): 435-50.
- [7] Aarsland D, Pålshagen S, Ballard CG, et al. Depression in Parkinson disease — epidemiology, mechanisms and management. *Nature Reviews Neurology* 2012, 8(1): 35-47.
- [8] Van Kessel SP, Frye AK, El-Gendy AO, et al. Gut bacterial tyrosine decarboxylases restrict levels of levodopa in the treatment of parkinson's disease. *Nature Communications* 2019, 10(1): 310-313.
- [9] Zhao JW, Guo YK. Effect of Bushen Huoxue Prescription Combined with Carbidopa and Levodopa Sustained-Release Tablets on Levels of Adiponectin, TNF- α and IL-6 in Serum and Cognitive Function of Patients with Parkinson Disease. *Journal of New Chinese Medicine* 2022, 54(8): 72-76.
- [10] Tang BC, Wang YT, Ren J. Basic information about memantine and its treatment of Alzheimer's disease and other clinical applications. *Ibrain* 2023, 9(3): 340-348.
- [11] Huang Q, Sun QJ, Guan JZ, et al. Effect of memantine hydrochloride combined with piribedil in the treatment of Parkinson's disease and its influence on cognitive function and motor function. *BMC Neurology* 2022, 13(5): 175.
- [12] Li J, Jin M, Wang L, et al. MDS clinical diagnostic criteria for Parkinson's disease in China. *Journal of Neurology* 2017, 264(3): 476-481.
- [13] Qu Y, Li XH, Sun YN, et al. Evaluation for Parkinson's Disease with Chinese Version of MDS-UPDRS and Original UPDRS. *Chinese Journal of Rehabilitation Theory and Practice* 2019, 25(8): 936-939.
- [14] Han L, Su XZ, Zhang ZY, et al. Effect of panlong needling at Jiaji (EX-B 2) on motor dysfunction in patients with Parkinson's disease of liver and kidney deficiency: a randomized controlled trial. *Zhongguo Zhen Jiu* 2022, 42(5): 493-7.
- [15] Moustafa AA, Chakravarthy S, Phillips JR, et al. Motor symptoms in Parkinson's disease: A unified framework. *Neuroscience & Biobehavioral Reviews* 2016, 68(9): 727-740.
- [16] Klamroth S, Steib S, Devan S, et al. Effects of exercise therapy on postural instability in parkinson disease: a meta-analysis. *Journal of Neurologic Physical Therapy* 2016, 40(1): 3-14.
- [17] Wang JL, Wu LL. Clinical Efficacy of Memantine Hydrochloride in the Treatment of Parkinson's Disease. *Journal of Aerospace Medicine* 2023, 34(3): 269-271.
- [18] Emre M, Tsolaki M, Bonuccelli U, et al. Memantine for patients with Parkinson's disease dementia or dementia with Lewy bodies: a randomised, double-blind, placebo-controlled trial. *The Lancet Neurology* 2010, 9(10): 969-77.
- [19] Jiang MF, Niu GM, Shi F, et al. The application of 1H MRS in Parkinson's disease. *Chinese Journal of Difficult and Complicated Cases* 2017, 16(10): 1010-1013.
- [20] Park SH, Choe JY, Kim SK, et al. Routine assessment of patient index data (RAPID3) and bath ankylosing spondylitis

disease activity index (BASDAI) scores yield similar information in 85 Korean patients with ankylosing spondylitis seen in usual clinical care. *Journal of Clinical Rheumatology* 2015, 21(6): 300-304.

[21] Zang CX, Bao XQ, Sun H, et al. The role of neuroinflammation-related regulatory targets in the treatment for Parkinson's disease. *Acta Pharmaceutica Sinica* 2016, 17(5): 677-683.

[22] Hickman S, Izzy S, Sen P, et al. Microglia in neurodegeneration. *Nature Neuroscience* 2018, 21(10): 1359-1369.

[23] Di Rita A, Strappazzon F. Mitophagy could fight Parkinson's disease through antioxidant action. *Reviews in the Neurosciences* 2019, 30(7): 729-742.

[24] Pizzino G, Irrera N, Cucinotta M, et al. Oxidative Stress:

Harms and Benefits for Human Health. *Oxidative Medicine and Cellular Longevity* 2017: 8416763.

[25] Dias V, Junn E, Mouradian MM. The role of oxidative stress in Parkinson's disease. *Journal of Parkinson's Disease* 2013; 3(4): 461-91.

[26] Qiao O, Zhang XY, Zhang Y, et al. Cerebralcare Granule® enhances memantine hydrochloride efficacy in APP/PS1 mice by ameliorating amyloid pathology and cognitive functions. *Chinese Medicine* 2021, 28(6): 47.

[27] Choi H, Hwang S, Cho H, et al. Memantine modulates neuroinflammation and motor coordination in a Parkinson's disease model. *Brain Research* 2025, 1869: 150034.

[28] Tang BC, Wang YT, Ren J. Basic information about memantine and its treatment of Alzheimer's disease and other clinical applications. *iBrain* 2023, 9(3): 340-348.