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Analysis of the Efficacy of Multi-Target Receptor Modulators Combined with Modified Electroconvulsive Therapy in BD Patients with Comorbid Multisystem Somatic Diseases

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Abstract: *Objective*: To evaluate the efficacy of multi-target receptor modulators combined with modified electroconvulsive therapy in BD patients with comorbid multisystem somatic diseases. *Methods*: A total of 68 BD patients with comorbid multisystem somatic diseases treated from May 2022 to May 2025 were selected as samples and randomly divided into groups by drawing lots. Group A received modified electroconvulsive therapy (MECT) combined with the multi-target receptor modulator quetiapine, while Group B received quetiapine alone. *Results*: Group A showed superior efficacy, Mini-Mental State Examination (MMSE) scores, Hamilton Depression Rating Scale (HAMD) scores, Bech-Rafaelsen Mania Rating Scale (BRMS) scores, and serum factor indicators compared to Group B, with $p < 0.05$; there was no significant difference in the adverse reaction rate between Group A and Group B, with $p > 0.05$. *Conclusion*: The combination of multi-target receptor modulators and modified electroconvulsive therapy can improve intellectual status, alleviate manic and depressive symptoms, and is highly effective and feasible in the treatment of BD patients with comorbid multisystem somatic diseases.

Keywords: Bipolar disorder; Quetiapine; MECT; MMSE

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1. Introduction

Bipolar Disorder (BD) with comorbid multisystem somatic diseases is characterized by recurrent episodes and is a common clinical affective disorder. During episodes, patients may experience alternating depressive and manic symptoms. Additionally, BD patients with comorbid multisystem somatic diseases often have cognitive impairments, manifested as decreased memory, difficulty concentrating, and reduced executive

function, necessitating early pharmacological intervention. When treating BD patients, quetiapine, a multi-target receptor modulator, is recommended for its significant antipsychotic effects. However, monotherapy with this drug has limited efficacy and requires combination with other treatment modalities. MECT is a modified version of traditional electroconvulsive therapy and can be used as an adjunctive treatment for severe BD patients with comorbid multisystem somatic diseases [1]. Based on this, this study explored the efficacy of MECT combined with quetiapine in 68 BD patients with comorbid multisystem somatic diseases from May 2022 to May 2025.

2. Materials and methods

2.1. Materials

A total of 68 BD patients with comorbid multisystem somatic diseases treated from May 2022 to May 2025 were selected as samples and randomly divided into groups by drawing lots. The baseline data of BD patients with comorbid multisystem somatic diseases in Group A were compared with those in Group B, with $p > 0.05$. See **Table 1**.

Table 1. Baseline data analysis table for BD patients with comorbid multisystem somatic diseases

Group	n	Gender (%)		Age (years)		Disease duration (months)	
		Male	Female	Range	Mean ± SD	Range	Mean ± SD
Group A	34	19 (55.88)	15 (44.12)	63–87	73.84 ± 4.29	1–9	4.56 ± 1.28
Group B	34	20 (58.82)	14 (41.18)	63–88	73.91 ± 4.31	1–8	4.61 ± 1.31
χ^2/t	-	0.0673		0.0502		0.1564	
p	-	0.7952		0.9601		0.8763	

2.2. Inclusion and exclusion

2.2.1. Inclusion criteria

(1) Meeting the criteria for BD as outlined in the third edition of the "Chinese Classification and Diagnostic Criteria of Mental Disorders" [2];
(2) Signing the informed consent form;
(3) Scoring ≥ 13 on the BRMS.

2.2.2. Exclusion criteria

(1) Individuals with severe suicidal tendencies;
(2) Those with a history of hypersensitivity to antipsychotic drugs;
(3) Patients with poor treatment compliance.

2.3. Treatment methods

(1) Group A

On the first day, patients received a single oral dose of 50 mg quetiapine once daily, followed by a gradual increase in the daily dose to 300–400 mg, administered over 4 weeks. Prior to MECT treatment, heart rate and blood pressure were regulated to 90 beats/min and 140/90 mmHg, respectively.

Subsequently, patients received an intravenous injection of 0.01 mg/kg atropine plus an intravenous bolus of 1.5 mg/kg propofol. Once the absence of eyelash reflex was observed in BD patients, an intravenous injection of 1 mg/kg succinylcholine chloride injection was administered. Throughout the procedure, patients were provided with pressurized oxygen via a mask. After medication, the patient's muscles relaxed completely. Electrodes were placed bilaterally on the brain, and the current was adjusted for impedance testing. Subsequently, MECT parameters were adjusted, with current, voltage, and stimulus frequency set to 0.9–1.2 A, 220 V, and 30–50 Hz, respectively. Each treatment session lasted 1–3 seconds, with three consecutive stimulations. Successful stimulation was defined by the patient exhibiting muscle tonus leading to clonus and then relaxation upon electrical stimulation. Stimulation was administered three times in the first week, twice in the second week, and once in weeks 3–4.

(2) Group B

On the first day, patients received a single oral dose of 50 mg quetiapine once daily, followed by a gradual increase in the daily dose to 300–400 mg, administered over 4 weeks.

2.4. Statistical analysis

SPSS 23.0 was used to process data on BD patients with comorbid multisystem somatic diseases. Count data for BD patients with comorbid multisystem somatic diseases were recorded as percentages and analyzed using the χ^2 test, while measurement data were recorded as mean ± standard deviation ($\overline{x} \pm s$) and analyzed using the *t*-test. Statistical significance was defined as $P < 0.05$.

3. Results

3.1. Efficacy analysis of BD with comorbid multisystem somatic diseases

The efficacy of BD treatment with comorbid multisystem somatic diseases was higher in Group A than in Group B, with $p < 0.05$. See **Table 2**.

Table 2. Efficacy analysis table for BD with comorbid multisystem somatic diseases (n, %)

Group	Markedly effective	Effective	Ineffective	Total effective rate
Group A (n = 34)	22 (64.71)	11 (32.35)	1 (2.94)	33 (97.06)
Group B (n = 34)	17 (50.00)	10 (29.41)	7 (20.59)	27 (79.41)
χ^2	-	-	-	5.1000
p	-	-	-	0.0239

3.2. Analysis of prognostic evaluation indicators for BD with comorbid multisystem somatic diseases

After treatment, the MMSE scores of BD patients with comorbid multisystem somatic diseases in Group A were higher than those in Group B, while the HAMD scores and BRMS scores were lower than those in Group B, with $p < 0.05$. See **Table 3**.

Table 3. Analysis table of prognostic evaluation indicators for bd with comorbid multisystem somatic diseases ($\overline{x} \pm s$)

Group	MMSE score (points)		HAMD score (points)		BRMS score (points)	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Group A (n = 34)	24.68 ± 3.42	29.72 ± 3.81	36.42 ± 5.43	20.66 ± 2.43	23.73 ± 4.11	10.18 ± 1.89
Group B (n = 34)	24.71 ± 3.39	26.25 ± 3.68	36.44 ± 5.47	25.73 ± 4.11	23.71 ± 4.09	14.33 ± 2.43
t	0.0363	3.8198	0.0151	6.1917	0.0201	7.8605
p	0.9711	0.0003	0.9880	0.0000	0.9840	0.0000

3.3. Analysis of serum factor indicators in BD patients with comorbid multisystem somatic diseases

After treatment, the serum factor indicators of BD patients with comorbid multisystem somatic diseases in Group A were superior to those in Group B, with $p < 0.05$. See **Table 4**.

Table 4. Analysis table of serum factor indicators in BD patients with comorbid multisystem somatic diseases ($\overline{x} \pm s$)

Group	TNF-α (ng/L)		IL-1β (g/L)		MDA (nmol/mL)		SOD (ng/L)	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Group A (n = 34)	101.88 ± 4.29	82.44 ± 2.42	91.42 ± 3.82	76.09 ± 2.19	7.54 ± 0.88	1.71 ± 0.43	96.28 ± 4.08	154.28 ± 6.72
Group B (n = 34)	101.91 ± 4.31	93.28 ± 3.29	91.38 ± 3.79	82.44 ± 2.88	7.59 ± 0.91	2.78 ± 0.62	96.33 ± 4.06	131.26 ± 5.33
t	0.0288	15.4762	0.0433	10.2338	0.2303	8.2690	0.0507	15.6496
p	0.9771	0.0000	0.9656	0.0000	0.8186	0.0000	0.9598	0.0000

3.4. Analysis of adverse reaction indicators in BD patients with comorbid multisystem somatic diseases

There was no significant difference in the adverse reaction rate between Group A and Group B of BD patients with comorbid multisystem somatic diseases, with $p > 0.05$. See **Table 5**.

Table 5. Analysis table of adverse reactions in BD patients with comorbid multisystem somatic diseases (n, %)

Group	Nausea and vomiting	Anorexia	Constipation	Lethargy	Incidence (%)
Group A (n = 34)	1 (2.94)	1 (2.94)	1 (2.94)	1 (2.94)	4 (11.76)
Group B (n = 34)	1 (2.94)	2 (5.88)	1 (2.94)	1 (2.94)	5 (14.71)
χ^2	-	-	-	-	0.1281
p	-	-	-	-	0.7205

4. Discussion

Bipolar disorder (BD) is a prevalent and severe mental disorder in clinical settings, characterized by

alternating episodes of mania and depression. The disease course is protracted and recurrent, impairing patients' social functioning and leading to suicidal ideation and behaviors. In recent years, the incidence of comorbid multisystem somatic diseases in BD patients has been increasing annually, becoming a critical factor affecting treatment prognosis and exacerbating disease burden [3]. The etiology of BD comorbid with multisystem somatic diseases is complex, and the specific pathogenesis remains unclear in clinical practice. It is generally believed that the onset of this disease is closely related to genetic predisposition factors, neurobiochemical disturbances, immune-inflammatory imbalances, and environmental stress factors. Genetic predisposition factors determine the innate propensity for disease onset in patients. Individuals carrying BD susceptibility genes inherently have potential abnormalities in neurotransmitter regulation, making them susceptible to manic or depressive episodes triggered by external stimuli. At the neurobiochemical level, imbalances in the secretion of monoamine neurotransmitters not only constitute the core pathological basis for the psychiatric symptoms of BD but also affect the function of various systems in the body through the neuro-immune-endocrine network, potentially inducing cardiovascular, digestive, endocrine, and other multisystem somatic diseases. Immune-inflammatory disturbances play a crucial mediating role in the development of comorbidities. BD patients often experience chronic inflammatory states, and abnormal activation of inflammatory factors can damage neuronal function while also affecting somatic tissues and organs, exacerbating the severity of somatic diseases. Environmental stress factors serve as triggers for the disease, disrupting the balance of the neuro-immune-endocrine system and promoting the comorbidity of BD and somatic diseases [4]. Patients with BD comorbid with multisystem somatic diseases exhibit more complex clinical symptoms. In addition to the typical alternating episodes of mania and depression, they also present with corresponding symptoms of the somatic systems, such as abnormal blood pressure and arrhythmia in the cardiovascular system, abdominal distension and decreased appetite in the digestive system, and blood glucose fluctuations and abnormal thyroid function in the endocrine system. Moreover, psychiatric symptoms and somatic symptoms interact and form a vicious cycle, leading to recurrent episodes of somatic diseases and even exacerbating patients' emotional fluctuations, triggering acute episodes of BD. Conversely, the psychiatric symptoms of BD can reduce patients' treatment compliance, affecting the therapeutic outcomes of somatic diseases and resulting in uncontrolled conditions for both, thereby increasing the difficulty of clinical treatment.

Quetiapine is a commonly used multi-target receptor modulator in the clinical treatment of BD. By regulating the activity of various neurotransmitter receptors in the brain, it improves the imbalance in neurotransmitter secretion and exerts a certain control over manic and depressive symptoms in BD patients, accumulating substantial practical experience in clinical applications. However, for BD patients comorbid with multisystem somatic diseases, the limitations of monotherapy with quetiapine are evident, and it fails to meet clinical treatment needs. Monotherapy with quetiapine has a slow onset of action. For patients experiencing acute manic or depressive episodes of BD, it cannot rapidly alleviate symptoms triggered by extreme emotions. Prolonged emotional deterioration can further exacerbate disturbances in the neuro-immune-endocrine network, inducing pathological damage to somatic diseases and even leading to acute episodes of somatic conditions. Monotherapy with quetiapine cannot simultaneously improve psychiatric and somatic symptoms. Its primary target sites are concentrated on neurotransmitter receptors, with limited regulatory effects on pathological states such as immune-inflammatory disturbances and oxidative stress imbalances present in comorbid BD patients. It cannot fundamentally break the vicious cycle between

psychiatric symptoms and somatic diseases, resulting in incomplete remission of psychiatric symptoms and recurrent somatic disease conditions in some patients after treatment, leading to suboptimal overall therapeutic outcomes [5]. Additionally, long-term monotherapy with quetiapine may induce various adverse reactions, such as weight gain, metabolic abnormalities, and cardiovascular discomfort, which can further increase the somatic burden of comorbid patients and even affect their treatment compliance, leading to treatment discontinuation.

Modified electroconvulsive therapy (MECT) is a physical treatment method that significantly reduces the adverse reactions of traditional electroconvulsive therapy while preserving its therapeutic efficacy by modifying electrode placement and controlling electrical stimulation dosage. It has been widely applied in the clinical treatment of severe mental disorders such as BD. MECT has a rapid onset of action. By inducing synchronous neuronal discharge in the brain through brief electrical stimulation, it can rapidly regulate abnormal activation states of brain neural circuits, improve neurotransmitter secretion balance, and effectively control acute manic and depressive symptoms in BD patients within a short period, creating favorable conditions for the treatment of somatic diseases. Based on this, adopting MECT in combination with the multi-target receptor modulator quetiapine to treat BD patients comorbid with multisystem somatic diseases can produce a synergistic therapeutic effect, compensating for many deficiencies of monotherapy with quetiapine and demonstrating significant treatment advantages. After MECT rapidly alleviates acute emotional symptoms of BD, quetiapine can continuously stabilize neurotransmitter levels through its multi-target regulatory effects, consolidate the therapeutic efficacy of MECT, reduce the recurrence of psychiatric symptoms, and simultaneously improve patients' cognitive and social functioning [6]. The synergistic action of the two can effectively regulate immune-inflammatory pathways in comorbid patients, inhibit excessive release of inflammatory factors, regulate oxidative stress balance, alleviate pathological damage to somatic tissues and organs, simultaneously improve somatic disease symptoms, and break the vicious cycle between psychiatric symptoms and somatic diseases. The synergistic effect of MECT and quetiapine can also reduce the dosage of single drugs, lower the risk of adverse reactions, and improve patients' treatment compliance. It is particularly suitable for BD patients comorbid with multisystem somatic diseases who have poor tolerance, providing a safer and more effective treatment option for clinical management of such complex cases.

The data in this article indicate that the therapeutic efficacy in BD patients comorbid with multisystem somatic diseases increased after treatment with MECT + quetiapine. The reasons for this are as follows: This study strictly adhered to clinical treatment guidelines for BD and comorbid somatic diseases, individually adjusting the quetiapine dosage and MECT treatment frequency and course based on the severity of patients' conditions, the type of somatic diseases, and their tolerance. This ensured the targeted and reasonable nature of the treatment plan, avoiding suboptimal efficacy due to improper dosages or non-standardized treatment. Simultaneously, a comprehensive efficacy evaluation system was established, adopting unified efficacy evaluation criteria to regularly assess improvements in patients' psychiatric and somatic symptoms, ensuring the objectivity and accuracy of efficacy evaluation results and truly reflecting the efficacy advantages of the combined treatment plan, resulting in a stable increasing trend in efficacy-related data [7]. The synchronous standardized intervention for patients' somatic diseases during the study also provided important guarantees for the increased efficacy, avoiding the impact of fluctuations in somatic disease conditions on the treatment outcomes of psychiatric symptoms.

Another set of data indicates that after treatment with MECT + quetiapine, the MMSE scores increased,

while the HAMD and BRMS scores decreased in BD patients comorbid with multisystem somatic diseases. The reasons for this are as follows: The MMSE score is primarily used to assess patients' cognitive function, including multiple dimensions such as attention, memory, and orientation. The HAMD score is used to evaluate the severity of depressive symptoms, and the BRMS score is used to assess the severity of manic symptoms. This study selected reasonable time points for score detection, enabling the timely capture of dynamic changes in patients' cognitive function and emotional states. Simultaneously, all scoring personnel underwent unified training and strictly adhered to scoring standards, reducing human assessment errors and ensuring the reliability of scoring data, allowing each scoring indicator to truly reflect the treatment outcomes. Analyzing the treatment mechanism, MECT can rapidly alleviate patients' acute emotional symptoms, reducing the inhibitory effect of emotional abnormalities on cognitive function and creating conditions for cognitive function recovery. Quetiapine gradually improves patients' cognitive function and emotional stability by regulating neurotransmitter balance through multi-target effects. The synergistic action of the two promotes an increase in MMSE scores and a decrease in HAMD and BRMS scores. Additionally, dynamic monitoring of patients' cognitive function and emotional states during treatment allows for timely adjustment of the treatment plan, avoiding cognitive impairment or emotional fluctuations during the treatment process and facilitating improvements in each scoring indicator.

Another set of data indicates that after treatment with MECT + quetiapine, the levels of TNF-α, IL-1β, MDA, and SOD improved in BD patients comorbid with multisystem somatic diseases. The reasons for this are as follows: TNF-α and IL-1β are important inflammatory factors, and their elevated levels indicate the presence of chronic inflammatory responses in the body. MDA is a marker of oxidative stress damage, and its elevated level reflects increased oxidative stress damage in the body. SOD is an important antioxidant enzyme in the body, and its elevated level indicates enhanced antioxidant capacity. This study strictly standardized the specimen collection process, selecting fasting venous blood as the detection specimen and promptly processing and storing it after collection to avoid factors such as specimen contamination and hemolysis affecting the detection results. Standardized detection methods and calibrated detection instruments were used to ensure the accuracy and repeatability of the detection results, truly reflecting changes in patients' immune-inflammatory and oxidative stress states. The synergistic action of MECT and quetiapine can effectively regulate the body's neuro-immune-endocrine network, inhibit excessive secretion of inflammatory factors, reduce the production of oxidative stress products, and enhance the body's antioxidant capacity, thereby promoting a decrease in TNF-α, IL-1β, and MDA levels and an increase in SOD levels. The effective exertion of this treatment mechanism results in significant improvements in immune-inflammatory and oxidative stress-related indicators.

The final set of data indicates that after treatment with MECT + quetiapine, the incidence of adverse reactions did not increase in BD patients comorbid with multisystem somatic diseases. The reasons for this are as follows: Based on patients' somatic disease conditions and tolerance, the quetiapine dosage and MECT treatment parameters were individually adjusted to reduce the occurrence of dosage-dependent adverse reactions. For example, for patients comorbid with cardiovascular diseases, the initial quetiapine dosage was appropriately reduced, and blood pressure and heart rate changes were closely monitored. For patients comorbid with digestive system diseases, the medication administration time was adjusted to reduce gastrointestinal adverse reactions.

5. Conclusion

In summary, BD patients comorbid with multisystem somatic diseases who received MECT + quetiapine treatment experienced suppression of inflammatory and oxidative stress responses, alleviation of manic and depressive symptoms, and optimization of cognitive function. Moreover, MECT demonstrated high safety and has promotional value.

Disclosure statement

The author declares no conflict of interest.

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