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Original Article

Mosapride combined with rebamipide demonstrates superior efficacy for treatment of chronic atrophic gastritis

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Abstract: Objective: To evaluate the therapeutic efficacy of combining mosapride with rebamipide for treatment of chronic atrophic gastritis (CAG). Methods: A total of 116 patients with confirmed CAG were enrolled. The control group (n=52) received mosapride alone, while the observation group (n=64) was treated with both mosapride and rebamipide. Therapeutic outcomes, adverse events, pathological scores, symptom relief time, and serological markers were compared between the groups. Univariate and multivariate analyses were also conducted to identify factors influencing treatment efficacy. Results: The observation group exhibited a significantly higher overall effective rate than the control group (P<0.05), with no significant difference in adverse events (P>0.05). Pathological scores were significantly lower in the observation group both compared to baseline and to the control group (P<0.05). Additionally, the observation group had greater improvements in all serological markers and a shorter duration to symptom relief (all P<0.05). Multivariate analysis identified smoking history (P=0.017, OR=4.318), alcohol consumption history (P=0.002, OR=6.327), and epidermal growth factor levels (P=0.044, OR=3.394) as independent risk factors for treatment response. Conclusion: The combination of mosapride and rebamipide offers superior efficacy in managing CAG without increasing adverse effects. It significantly improves pathological conditions, expedites symptom resolution, and enhances gastric mucosal biomarker profiles, supporting its broader clinical application.

Keywords: Mosapride, rebamipide, chronic atrophic gastritis, efficacy, influencing factors

Introduction

Chronic atrophic gastritis (CAG), recognized as a precancerous lesion of gastric cancer, is fundamentally a chronic inflammatory condition [1]. It is characterized by the atrophy and loss of gastric mucosal epithelium and glands, often accompanied by intestinal or pyloric gland metaplasia. The condition is closely associated with *Helicobacter pylori* (HP) infection [2]. Epidemiological data suggest that the prevalence of CAG in the general population is approximately 25.0%, with HP-positive individuals exhibiting a 2.4-fold higher risk compared to HP-negative individuals [3]. Clinically, CAG typically manifests as epigastric distension, pain, and anorexia. However, most patients are asymptomatic in the early stages and may only develop non-specific symptoms - such as upper gastrointestinal discomfort, autoimmune manifestations, or pernicious anemia-during advanced stages [4, 5]. Current conventional therapies offer suboptimal efficacy and safety profiles, highlighting the need for improved treatment strategies to better alleviate symptoms and enhance clinical outcomes [6].

Mosapride, a 5-hydroxytryptamine 4 (5-HT4) receptor agonist, has shown clinical safety in the treatment of various gastrointestinal disorders [7, 8]. Its therapeutic effect is primarily mediated through the stimulation of acetylcholine release at parasympathetic nerve endings, which enhances esophageal motility and promotes gastric emptying [9]. However, some studies have reported that mosapride does not provide additional symptom relief in patients with gastroesophageal reflux disease and may negatively affect treatment adherence due to discomfort [10].

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Rebamipide, an amino acid derivative, enhances gastric mucosal defense by stimulating the production of endogenous prostaglandins and inhibiting the generation of free radicals. It also exhibits anti-inflammatory effects by suppressing cytokine production and neutrophil activation [11, 12]. A recent systematic review and meta-analysis confirmed the clinical efficacy and safety of rebamipide in the treatment of CAG [13].

Despite the individual therapeutic potentials of mosapride and rebamipide, studies investigating their combined use for CAG remain limited. Therefore, this study aims to address this gap by systematically evaluating the efficacy of combination therapy and identifying key factors influencing treatment response, thereby offering more effective clinical strategies for managing CAG.

Materials and methods

General patient information

This retrospective study included 116 patients diagnosed with CAG, all of whom were admitted to the Wuxing District People's Hospital of Huzhou between August 2022 and December 2024. Patients were divided into two groups based on treatment regimen: the control group (n=52) received mosapride monotherapy, while the observation group (n=64) was treated with a combination of mosapride and rebamipide. Ethical approval for this study was obtained from the Ethics Committee of the First Affiliated Hospital of Huzhou University.

Inclusion criteria: (1) Diagnosis met the established criteria for CAG [14]; (2) Patients presented with symptoms such as nausea, vomiting, gastric distension, decreased appetite, and recurrent epigastric pain lasting more than six months; (3) Helicobacter pylori (HP) status was confirmed using either a $^{13}C/^{14}C$ urea breath test or a rapid urease test of the gastric mucosa. HP-positive patients had completed eradication therapy and were confirmed negative upon retesting before enrollment; (4) No use of proton pump inhibitors (PPIs), H2 receptor antagonists, bismuth compounds, or other gastric mucosal protective agents within two weeks prior to enrollment; (5) Complete and accurate clinical data were available.

Exclusion criteria: (1) Pregnant or lactating women; (2) Patients with ultrasound or radiographic evidence of metabolic or organic lesions in the liver, gallbladder, pancreas, or intestines; (3) History of peptic ulcer, prior abdominal surgery, or malignancy; (4) Diagnosed psychiatric or psychological disorders; (5) Coexisting gastric, hepatobiliary, pancreatic, or intestinal diseases; (6) Comorbid metabolic or autoimmune diseases, or severe dysfunction of the heart, kidneys, or lungs; (7) Recent use of medications or history of surgery; (8) History of alcohol or substance abuse, or psychological abnormalities.

Medication regimens

The control group received mosapride (Shanghai Jingfeng Biological Science and Technology Co., Ltd., JF1050470) at a dose of 5 mg orally, three times daily before meals. The observation group received the same mosapride regimen, in combination with rebamipide (Beijing Wobison Technology Co., Ltd., VS19573-25g), taken at 0.1 g per dose, three times daily. Both groups were treated continuously for three months.

A comparative analysis was conducted to evaluate therapeutic efficacy, adverse reactions (including dry mouth, fatigue, headache, and gastrointestinal symptoms), pathological scores (gastric mucosal inflammation score and pathological grading score), and serological markers [gastrin 17 (G-17), pepsinogen I (PGI), and epidermal growth factor (EGF)] between the two groups.

Therapeutic efficacy

Therapeutic efficacy was classified as follows.

Markedly effective: Clinical symptoms completely or nearly resolved; gastroscopy indicated conversion from atrophic gastritis to superficial gastritis, with a significant reduction in pale submucosal areas, restored vascular visibility, and predominantly pink mucosa.

Effective: Clinical symptoms improved; gastroscopy showed improvement in mucosal atrophy, reduction in pale areas and vascular visibility, and partial recovery of orange-red mucosal coloration.

Ineffective: Clinical symptoms remained unchanged or worsened, with no significant improvement observed in histological or gastroscopic findings.

The total effective rate was calculated as the proportion of cases classified as markedly effective or effective out of the total number of cases.

Adverse reactions

The incidence of adverse effects (dry mouth, fatigue, headache, and gastrointestinal symptoms) was recorded in both groups, and the overall incidence rate was calculated.

Pathological scoring

Gastric mucosal inflammation scores and corresponding pathological grading scores were compared between the two groups before and after three months of treatment.

The gastric mucosal inflammation score ranges from 0 to 3: 0 indicates no inflammation; 1, mild; 2, moderate; and 3, severe inflammation.

The pathological grading score ranges from 0 to 4: 0: no erosions; 1: ≤2 erosions confined to one area; 2: 3-5 erosions in a single area; 3: <6 erosions across two regions; 4: >3 erosion areas and >10 erosions in total.

Symptom relief time

The time to relief of clinical symptoms (pain, bloating, and general recovery) was compared between the two groups.

Serological markers

A 5 mL fasting peripheral venous blood sample was collected before and after treatment. Serum was isolated via centrifugation. Levels of G-17 and PGI were measured using enzyme-linked immunosorbent assay (ELISA; Shanghai Qincheng Biotechnology Co., Ltd., QC13978-A; Shanghai Center Biology Science and Technology Co., Ltd., QY-SE1357, QY-SE0183). EGF levels were assessed using radioimmunoassay.

Statistical analysis

Continuous variables were expressed as mean ± standard error of the mean (SEM). Between-group comparisons were conducted using the independent-samples t-test, and within-group (pre- vs. post-treatment) comparisons were analyzed using the paired t-test. Categorical variables were expressed as rates (percentages), and intergroup comparisons were performed using the chi-square (χ^2) test. All statistical analyses were conducted using SPSS version 20.0 (Baiao Yijie [Beijing] Technology Co., Ltd.). A *P*-value <0.05 was considered significant.

Results

Comparative analysis of general characteristics

There were no significant differences between the control and observation groups in terms of gender, age, disease duration, smoking history, alcohol consumption history, or family medical history (all P>0.05) (**Table 1**).

Comparative analysis of therapeutic efficacy

The overall treatment efficacy rate was 75.00% in the control group and 89.07% in the observation group. The observation group showed a significantly higher efficacy rate compared to the control group (P<0.05) (**Table 2**).

Comparative analysis of adverse reactions

The incidence of adverse reactions-specifically dry mouth, fatigue, headache, and gastrointestinal symptoms-was 0, 1, 1, and 3 cases in the control group, and 1, 0, 1, and 2 cases in the observation group, respectively. There was no significant difference in the overall incidence of adverse reactions between the two groups (P>0.05) (**Table 3**).

Comparative analysis of pathological scores

Before treatment, no significant differences were observed between the two groups in either the gastric mucosal inflammation score or the pathological grading score (both P> 0.05). After treatment, both scores decreased significantly in both groups, with the observation group exhibiting significantly lower post-treatment scores compared to the control group (all P<0.05) (**Figure 1**).

Table 1. Comparative analysis of general characteristics

Indicator	n	Control group (n=52)	Observation group (n=64)	t/χ^2	P
Sex				1.159	0.282
Male	65	32 (61.54)	33 (51.56)		
Female	51	20 (38.46)	31 (48.44)		
Age (years)	116	48.50±5.23	49.30±8.56	0.590	0.556
Disease course (years)	116	2.54±0.90	2.38±1.09	0.849	0.398
Smoking history				0.402	0.526
Without	64	27 (51.92)	37 (57.81)		
With	52	25 (48.08)	27 (42.19)		
Alcohol consumption history				1.392	0.238
Without	78	32 (61.54)	46 (71.88)		
With	38	20 (38.46)	18 (28.13)		
Family medical history				0.786	0.375
Without	94	44 (84.62)	50 (78.13)		
With	22	8 (15.38)	14 (21.88)		

Table 2. Comparative analysis of therapeutic efficacy

Indicator	Control group (n=52)	Observation group (n=64)	χ^2	P
Markedly effective	22 (42.31)	38 (59.38)		
Effective	17 (32.69)	19 (29.69)		
Ineffective	13 (25.00)	7 (10.93)		
Overall efficacy	39 (75.00)	57 (89.07)	3.976	0.046

Table 3. Comparative analysis of adverse reactions

Indicator	Control group (n=52)	Observation group (n=64)	χ^2	P
Dry mouth	0 (0.00)	1 (1.56)		
Fatigue	1 (1.92)	0 (0.00)		
Headache	1 (1.92)	1 (1.56)		
Gastrointestinal reactions	3 (5.77)	2 (3.13)		
total	5 (9.62)	4 (6.25)	0.454	0.500

Comparative analysis of clinical symptom relief time

Clinical symptom relief times, including time to pain relief, bloating resolution, and overall recovery, were significantly shorter in the observation group compared to the control group (all P<0.05), indicating better symptom management with the combination therapy (**Table 4**).

Comparative analysis of serological markers

Before treatment, there were no significant differences in the levels of G-17, PGI, or EGF between the two groups (all P>0.05). After treatment, G-17 and PGI levels significantly increased, while EGF levels significantly decreased in both groups (all P<0.05). Post-treatment, the observation group showed significantly higher G-17 and PGI levels, and significantly lower EGF levels, compared to the control group (all P<0.05) (**Figure 2**).

Analysis of factors influencing therapeutic efficacy in CAG patients

Univariate analysis identified smoking history (P=0.013), alcohol consumption history (P< 0.001), G-17 level (P=0.041), EGF level (P=0.046), and treatment regimen (P=0.046) as factors significantly associated with treatment efficacy (P<0.05). These variables were subsequently included in a binary logistic regression analysis, where therapeutic efficacy was set as the dependent variable. The analysis identified smoking history (P=0.017, OR=4.318), alcohol consumption history (P=0.002, OR=6.327), and EGF level (P=0.044, OR=3.394) as independent risk factors significantly influencing treatment outcomes (P<0.05) (**Tables 5**-**7**).

Discussion

Chronic atrophic gastritis (CAG), a recognized precancerous lesion of the gastric epithelium, carries the potential for malignant transforma-

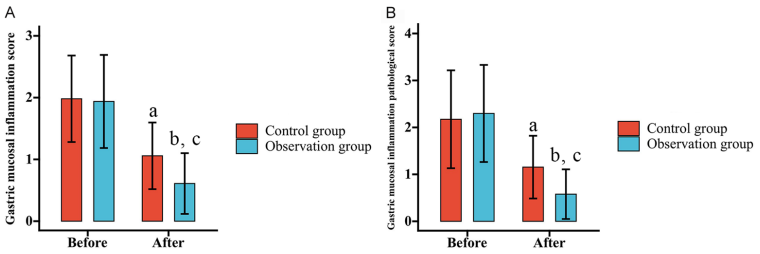

Figure 1. Comparative analysis of pathological scores. A. Gastric mucosal inflammation scores of the two groups before and after treatment. B. Gastric mucosal inflammation pathological scores before and after medication in the two groups. Note: [a]P<0.05, [b]P<0.01, when compared to the pre-treatment values; [c]P<0.05, when compared to the control group.

Table 4. Comparative analysis of clinical symptom relief time

Indicator	Control group (n=52)	Observation group (n=64)	t	P
Pain alleviation time (d)	4.69±2.07	2.45±1.36	6.999	<0.001
bloating resolution time (d)	3.27±1.43	1.36±0.76	9.209	<0.001
Recovery time (d)	11.02±3.81	9.44±2.79	2.576	0.011

tion into upper gastrointestinal cancers, including gastric, gastroesophageal junction, and esophageal cancers [15, 16]. However, current treatment options lack effective maintenance therapies, highlighting the urgent need for more effective treatment [17].

Mosapride enhances gastric motility by promoting acetylcholine release and reduces gastric mucosal damage by neutralizing excessive gastric acid. However, it lacks mucosal reparative properties, which limits its therapeutic efficacy as monotherapy [18, 19]. In contrast, rebamipide, a gastric mucosal protective agent, strengthens the mucosal barrier and improves mucosal blood flow, thereby compensating for mosapride's shortcomings [20].

Our study demonstrated that the combination of mosapride and rebamipide significantly improved overall therapeutic efficacy, with a response rate of 89.07% in the observation group compared to 75.00% in the control group. The concurrent administration of these agents appears to exert a synergistic effect, enhancing clinical outcomes. This finding is consistent with the results reported by Kang et al. [21], who observed that rebamipide combined with nizatidine improved mucosal erosion healing and alleviated gastrointestinal symptoms in erosive gastritis.

Furthermore, the safety profiles of both treatments were comparable, with no significant increase in adverse events such as dry mouth, fatigue, headache, or gastrointestinal reactions in the combination group. Similar findings were reported by Wang et al. [22], who demonstrated that rebamipide improved PPI-induced ulcer healing without serious adverse effects following endoscopic submucosal dissection.

The combination therapy also significantly reduced gastric mucosal inflammation, slowed pathological progression, and accelerated symptom relief compared to mosapride alone. These findings align with those of Han et al. [23], who reported that rebamipide effectively improved clinical symptoms, mucosal lesions, and histological grade in patients with chronic gastritis.

Growing evidence suggests that low serum levels of G-17 and PGI are closely associated with atrophic gastritis in the antrum and corpus, respectively, while EGF plays a critical role in the proliferation and differentiation of gastric epithelial cells [24, 25]. In our study, the combination therapy significantly increased G-17 and PGI levels and reduced EGF levels post-treatment.

Univariate analysis identified smoking history, alcohol consumption history, G-17 level, EGF level, and treatment modality as factors associated with therapeutic efficacy. Multivariate logistic regression further revealed that smoking history, alcohol consumption history, and elevated EGF level were independent predictors of poorer therapeutic response. The underlying mechanisms are likely multifactorial. Smoking and alcohol use may exacerbate gastric mucosal damage through reduced blood flow, increased acid secretion, bile reflux, and disruption of intestinal microbiota. Elevated EGF may promote abnormal epithelial proliferation, aggravating inflammation and injury [26-28].

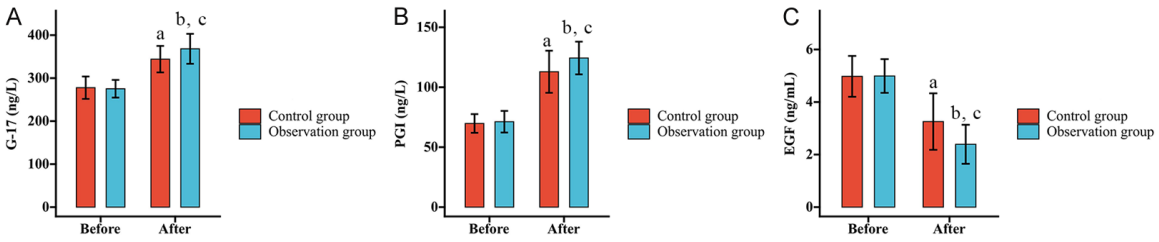

Figure 2. Comparative analysis of serological markers. A. G-17 levels of the two groups before and after treatment. B. PGI levels of the two groups before and after treatment. C. EGF levels of the two groups before and after treatment. Note: [a]P<0.05, [b]P<0.01, when compared to the pre-treatment values; [c]P<0.05, when compared to the control group. G-17, gastrin 17; PGI, pepsinogen I; EGF, epidermal growth factor.

Table 5. Univariate analysis of factors influencing therapeutic efficacy in patients with chronic atrophic gastritis

Indicator	n	Ineffective (n=20)	Effective (n=96)	χ^2	P
Sex				0.788	0.375
Male	65	13 (65.00)	52 (54.17)		
Female	51	7 (35.00)	44 (45.83)		
Age (years)				0.519	0.471
<50	67	13 (65.00)	54 (56.25)		
≥50	49	7 (35.00)	42 (43.75)		
Disease course (years)				0.050	0.824
<3	67	12 (60.00)	55 (57.29)		
≥3	49	8 (40.00)	41 (42.71)		
Smoking history				6.192	0.013
Without	64	6 (30.00)	58 (60.42)		
With	52	14 (70.00)	38 (39.58)		
Alcohol consumption history				11.405	<0.001
Without	78	7 (35.00)	71 (73.96)		
With	38	13 (65.00)	25 (26.04)		
Family medical history				0.573	0.449
Without	94	15 (75.00)	79 (82.29)		
With	22	5 (25.00)	17 (17.71)		
G-17 (ng/L)				4.180	0.041
<270	46	12 (60.00)	34 (35.42)		
≥270	70	8 (40.00)	62 (64.58)		
PGI (ng/L)				3.542	0.060
<270	59	14 (70.00)	45 (46.88)		
≥270	57	6 (30.00)	51 (53.13)		
EGF (ng/mL)				3.976	0.046
<5	64	7 (35.00)	57 (59.38)		
≥5	52	13 (65.00)	39 (40.63)		
Treatment modality				3.976	0.046
Mosapride	52	13 (65.00)	39 (40.63)		
Mosapride + rebamipide	64	7 (35.00)	57 (59.38)		

Note: G-17, gastrin 17; PGI, pepsinogen I; EGF, epidermal growth factor.

Our findings underscore the superior efficacy of mosapride-rebamipide combination therapy in CAG treatment and identify key risk factors influencing treatment response. These results

Table 6. Assignments of clinical and laboratory factors

Variable	Variable	Assignment
Smoking history	X1	Without =0, with =1
Alcohol consumption history	X2	Without =0, with =1
G-17 (ng/L)	X3	≥270=0, <270=1
EGF (ng/mL)	X4	≥5=0, <5=1
Treatment modality	X5	Mosapride + rebamipide =0, mosapride =1
Therapeutic efficacy	Y	Effective =0, ineffective =1

Note: G-17, gastrin 17; EGF, epidermal growth factor.

Table 7. Multivariate analysis of factors influencing therapeutic efficacy in patients with chronic atrophic gastritis

Variable	β	SE	Wald	P	OR	95% CI
Smoking history	1.463	0.612	5.716	0.017	4.318	1.302-14.325
Alcohol consumption history	1.845	0.604	9.329	0.002	6.327	1.937-20.670
G-17 (ng/L)	-0.895	0.575	2.420	0.120	0.409	0.132-1.262
EGF (ng/mL)	1.222	0.607	4.059	0.044	3.394	1.034-11.144
Treatment modality	-0.968	0.591	2.682	0.101	0.380	0.119-1.210

Note: G-17, gastrin 17; EGF, epidermal growth factor.

provide strong support for the combination therapy as personalized treatment.

However, this study had several limitations. First, the absence of long-term follow-up data prevented evaluation of sustained therapeutic effects. Future studies should extend the observation period and include prognosis-related analyses. Second, the lack of inflammatory biomarkers limited the assessment of the treatment's anti-inflammatory mechanisms. Incorporating such indicators in future research could provide deeper insight into its immunomodulatory actions. Third, gut microbiota profiling was not performed; including microbial analysis may help clarify whether therapeutic benefits are partly mediated by microbial regulation. Finally, due to the limited sample size, neither internal (e.g., cross-validation) nor external validation using independent cohorts was feasible. Large-scale, prospective studies are needed to confirm the generalizability and clinical utility of our findings.

In summary, the combination of mosapride and rebamipide significantly improved treatment efficacy in patients with CAG. This approach is safe, mitigates inflammation-driven pathologic progression, accelerates symptom relief, and improves gastric mucosa-related biomarkers, particularly G-17, PGI, and EGF. Moreover, smoking history, alcohol consumption history, and elevated EGF levels were identified as significant risk factors worsening therapeutic outcomes. These findings highlight the need for close monitoring and lifestyle-based interventions-such as smoking cessation and alcohol abstinence-to optimize treatment efficacy and achieve a favorable clinical outcome.

Disclosure of conflict of interest

None.

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