

Clinical Experience of Digestive Internal Medicine Treatment for Patients with Chronic Atrophic Gastritis

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Abstract:

Objective: To investigate the treatment methods and therapeutic effects of digestive internal medicine for patients with chronic atrophic gastritis. **Methods:** Sixty patients with chronic atrophic gastritis admitted to our hospital from January 2022 to December 2023 were selected. They were divided into control group (n=30) receiving a triple therapy of furazolidone, levofloxacin combined with omeprazole, and observation group (n=30) receiving a triple therapy of furazolidone, levofloxacin combined with rabeprazole. The levels of inflammatory factors [interleukin-6 (IL-6), interleukin-10 (IL-10), tumor necrosis factor- α (TNF- α)] before and after treatment, clinical symptom scores (gastric acid, gastric pain, bloating, loss of appetite), disappearance time of clinical symptoms (epigastric pain, bloating and belching, nausea and vomiting), clinical scores (pathological histology grading, gastroscopy and pathological efficacy, clinical symptom scores), serum gastrin (GAS), endothelin (ET) levels, overall clinical efficacy, and incidence of adverse symptoms were compared between the two groups. **Results:** Before treatment, there was no statistically significant difference in the levels of IL-6, IL-10, and TNF- α between the two groups ($P>0.05$). After treatment, the levels of IL-6, IL-10, and TNF- α in the observation group were superior to those in the control group, and the difference was statistically significant ($P<0.05$). Before treatment, there was no statistically significant difference in the scores of gastric acid, gastric pain, bloating, and loss of appetite between the two groups ($P>0.05$). After treatment, the scores of gastric acid, gastric pain, bloating, and loss of appetite in the observation group were lower than those in the control group, and the difference was statistically significant ($P<0.05$). The disappearance time of epigastric pain, bloating and belching, nausea and vomiting in the observation group was shorter than that in the control group. The pathological histology grading and clinical symptom scores in the observation group were lower than those in the control group, and the gastroscopy and pathological efficacy scores in the observation group were higher than those in the control group, with statistically significant differences ($P<0.05$). The GAS level in the observation group was higher than that in the control group, and the ET level in the observation group was lower than that in the control group, with statistically significant differences ($P<0.05$). The overall clinical efficacy in the observation group was higher than that in the control group, and the difference was statistically significant ($P<0.05$). The incidence of adverse reactions in the observation group was lower than that in the control group, but there was no

statistically significant difference between the two groups ($P>0.05$). Conclusion: Triple therapy with furazolidone, levofloxacin combined with rabeprazole can improve the clinical efficacy of patients with chronic atrophic gastritis, promote symptom relief, and demonstrate good safety, which is worthy of promotion.

Keywords:

Chronic Atrophic Gastritis, Digestive Internal Medicine, Triple Therapy, Therapeutic Effect, Safety

1. Introduction

Chronic atrophic gastritis is a common disease in modern clinical digestive internal medicine, mainly caused by atrophic lesions of the gastric mucosa in patients. The occurrence of chronic atrophic gastritis usually develops gradually based on chronic superficial gastritis and atrophic gastritis, and the cure rate for this type of disease is relatively high. The clinical symptoms are not obvious in the early stage of the disease, and the affected area is relatively wide after the onset [1]. In recent years, the number of patients with chronic atrophic gastritis has gradually increased. After the occurrence of chronic atrophic gastritis, patients may experience varying degrees of decreased appetite, nausea, vomiting, and other related reactions, which seriously affect their daily life and dietary habits. If timely and effective measures are not taken to treat patients, there is a certain probability of developing gastric cancer, posing a threat to their life. Therefore, it is necessary to use medication for effective treatment in clinical practice. Currently, drug therapy is generally adopted in the clinical treatment of chronic atrophic gastritis. When using drug therapy, it is necessary to combine it with the actual physical condition of the patient, apply drugs rationally for targeted treatment, and ensure the safety of medication. This article aims to analyze the application effect of triple therapy in digestive internal medicine and present the following research [2].

2. Data and Methods

2.1. General Information

Selecting 60 patients with chronic atrophic gastritis admitted to our hospital from January 2022 to December 2023, they were grouped using a random number table method. The control group ($n=30$) received a triple therapy of furazolidone, levofloxacin, and omeprazole. The number of male and female patients in the control group was 18 and 12, respectively, with an age range of 55-86 years and an average age of (63.34 ± 4.68) years. The observation group ($n=30$) received a triple therapy of furazolidone, levofloxacin, and rabeprazole. The number of male and female patients in the observation group was 16 and 14, respectively, with an age range of 53-87 years and an average age of (63.10 ± 4.82) years. There were no significant differences in general information between the two groups ($P>0.05$), indicating comparability [3].

Inclusion criteria: (1) Diagnosed with chronic atrophic gastritis through comprehensive clinical diagnosis; (2) Complete clinical data available; (3) No prior treatment with other drugs before the study; (4) Patients and their families were informed about the study and signed the consent form [4].

Exclusion criteria: (1) Accompanied by cerebrovascular or cardiovascular diseases; (2) Accompanied by renal or hepatic diseases; (3) Presence of endocrine disorders; (4) Presence of coagulation system abnormalities; (5) History of allergy to the study drugs; (6) Accompanied by psychiatric disorders; (7) Presence of other digestive system-related diseases.

2.2. Methods

Control Group: Administered a triple therapy of furazolidone, levofloxacin, and omeprazole. Dosage instructions include: Furazolidone: Oral administration, 10mg per dose, three times a day; Levofloxacin: Oral administration, 0.2g per dose, twice a day; Omeprazole: Oral administration, 20mg per dose, twice a day, for a duration of 14 days [5].

Observation Group: Administered a triple therapy of furazolidone, levofloxacin, and rabeprazole. The usage of furazolidone and levofloxacin is the same as in the control group, while rabeprazole is administered orally, 20mg per dose, twice a day, for a duration of 14 days [6].

2.3. Observation Indicators

(1) Levels of inflammatory factors (Interleukin-6 (IL-6), Interleukin-10 (IL-10), Tumor Necrosis Factor-alpha (TNF- α)) before and after treatment: Patients fasted for 10 hours and had venous blood collected on the following morning. The collected blood sample (5ml) was centrifuged at 3000r/min for ten minutes to obtain serum for enzyme-linked immunosorbent assay.

(2) Clinical symptom scores before and after treatment (gastric acid, gastric pain, bloating, loss of appetite): Each symptom was scored as 0, 2, 4, or 6, with higher scores indicating more severe clinical symptoms [7].

(3) Time to disappearance of clinical symptoms (epigastric pain, bloating belching, nausea and vomiting) and clinical scores (pathological histological grading, gastroscopy and pathological efficacy, clinical symptom scores).

(4) Levels of serum gastrin (GAS) and endothelin (ET): Tested using the same method as for inflammatory factors.

(5) Overall clinical effective rate: Remarkable effect: all clinical symptoms and related signs of the patient disappeared or improved significantly. Effective: all clinical symptoms and vital signs of the patient improved. Ineffective: no improvement in all clinical symptoms and vital signs. The overall clinical effective rate = (Remarkable effect + Effective) / total number of cases in the group $\times$ 100%.

(6) Incidence of adverse symptoms: including abdominal distension, nausea and vomiting, epigastric obstruction, and eructation.

2.4. Statistical Analysis

Statistical analysis was conducted using SPSS 20.0 software. Mean $\pm$ standard deviation ($\pm$ s) was used to represent quantitative data, t-test for parametric data, percentage (%) for count data, and chi-square test (χ^2) for count data. A statistically significant difference was considered when $P < 0.05$ between the two groups [8].

3. Results

3.1. Comparison of levels of inflammatory factors before and after treatment

Before treatment, there was no statistically significant difference in the levels of IL-6, IL-10, and TNF- α between the two groups ($P>0.05$). After treatment, the levels of IL-6, IL-10, and TNF- α in the observation group were superior to those in the control group, with a statistically significant difference ($P<0.05$), as shown in Table 1.

Table 1. Comparison of levels of inflammatory factors before and after treatment ($\pm s$).

group	number	IL-6 (ng/L)		IL-10 (ng/L)		TNF- α (ng/L)	
		before treatment	after treatment	before treatment	after treatment	before treatment	after treatment
observation group	30	27.65 $\pm$ 3.18	34.84 $\pm$ 4.08	41.13 $\pm$ 3.06	50.83 $\pm$ 4.10	4.13 $\pm$ 1.05	6.83 $\pm$ 2.08
matched group	30	27.49 $\pm$ 3.13	41.67 $\pm$ 7.27	41.08 $\pm$ 3.10	71.70 $\pm$ 5.30	4.10 $\pm$ 1.01	12.71 $\pm$ 1.31
t	-	0.196	4.487	0.063	17.059	0.113	13.102
P	-	0.845	0.000	0.950	0.000	0.911	0.000

3.2. Comparison of clinical symptom scores before and after treatment

Before treatment, the scores of gastric acid, gastric pain, bloating, and loss of appetite in both groups were compared, with no statistically significant difference ($P>0.05$). After treatment, the scores of gastric acid, gastric pain, bloating, and loss of appetite in the observation group were lower than those in the control group, showing a statistically significant difference ($P<0.05$), as shown in Table 2.

Table 2. Comparison of clinical symptom scores before and after treatment ($\pm s$).

group	number	gastric acid		stomachache		gasteremphraxis		poor appetite	
		before treatment	after treatment	before treatment	after treatment	before treatment	after treatment	before treatment	after treatment
observation group	30	4.16 $\pm$ 1.05	0.82 $\pm$ 0.06	4.26 $\pm$ 1.15	0.83 $\pm$ 0.08	4.12 $\pm$ 0.86	0.77 $\pm$ 0.08	4.35 $\pm$ 0.94	0.81 $\pm$ 0.06
matched group	30	4.14 $\pm$ 1.01	1.69 $\pm$ 0.29	4.29 $\pm$ 1.16	1.72 $\pm$ 0.33	4.19 $\pm$ 0.89	1.46 $\pm$ 0.21	4.36 $\pm$ 0.98	1.66 $\pm$ 0.32
t	-	0.071	15.258	0.095	13.609	0.291	15.920	0.038	13.562
P	-	0.944	0.000	0.925	0.000	0.772	0.000	0.970	0.000

3.3. Comparison of time to disappearance of clinical symptoms and clinical scores

The time for the disappearance of epigastric pain, bloating belching, nausea, and vomiting in the observation group was shorter than that in the control group. The pathological histological grading and clinical symptom scores in the observation group were lower than those in the control group, and the gastroscopy and pathological efficacy scores in the observation group were higher than those in the control group, showing a statistically significant difference ($P<0.05$), as shown in Table 3.

Table 3. Comparison of clinical symptom resolution time and clinical score ($\pm s$).

group	number	Disappearance time of epigastric	Eructation time (d)	Nausea and vomitin	Histopathological grade	Gastroscopy and Pathologi	Clinical symptom score
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		pain (d)		g resoluti on time (d)		cal effect (minutes)	(score)
observati on group	30	4.24±1.25	5.37±2.0 6	5.97±1. 84	2.01±0.51	2.52±0.14	3.30±0. 11
matched group	30	6.75±1.32	8.68±1.9 7	8.97±2. 13	4.88±0.55	0.43±0.09	7.60±1. 15
t	-	7.562	6.360	5.838	20.958	68.781	20.387
P	-	0.000	0.000	0.000	0.000	0.000	0.000

Comparison of Serum Gastrin and Endothelin Levels

The GAS levels in the observation group of patients (182.69±13.56 g/L) were higher than those in the control group (145.13±12.48 g/L). The ET levels in the observation group (61.03±5.29 g/L) were lower than those in the control group (73.49±6.58 g/L). The differences were statistically significant ($P < 0.05$).

Comparison of Overall Clinical Efficacy

The observation group: 96.7% (29/30), with 19 cases of significant effectiveness, 10 cases of effectiveness, and 1 case of ineffectiveness. The control group: 73.3% (22/30), with 15 cases of significant effectiveness, 7 cases of effectiveness, and 8 cases of ineffectiveness. The overall clinical efficacy in the observation group was higher than that in the control group, and the difference was statistically significant ($P < 0.05$).

Comparison of Adverse Reaction Incidence

The observation group: 10.0% (3/30), with 1 case each of abdominal distension, nausea and vomiting, and epigastric stuffiness. The control group: 16.67% (5/30), with 2 cases of abdominal distension, 1 case each of nausea and vomiting, epigastric stuffiness, and belching. The incidence of adverse reactions in the observation group was lower than that in the control group, but the difference between the two groups was not statistically significant ($P > 0.05$).

4. Conclusions

Chronic atrophic gastritis is a common disease in modern clinical gastroenterology. The onset of this disease is slow, and early clinical symptoms are not characteristic, with some patients having no symptoms at all, which can easily be overlooked or misdiagnosed [9]. By the time of definite diagnosis, the optimal treatment opportunity may have been missed, leading to severe atrophy and even precancerous lesions, which increase the difficulty of clinical treatment and affect the prognosis. The etiology of chronic atrophic gastritis has not been confirmed yet in clinical practice, but it is believed to be the result of multiple factors, such as *Helicobacter pylori* infection, abnormal vascular active factors, autoimmune system factors, improper diet, and infections. With the improvement in people's quality of life and pace of life in modern society, the incidence and number of patients with chronic atrophic gastritis have been increasing, and the main affected population is becoming younger, greatly affecting the patients' physical health and quality of life [10].

There is no consensus on the clinical treatment plan for chronic atrophic gastritis patients. Generally, a triple therapy regimen is adopted, which commonly includes levofloxacin, furazolidone, and omeprazole. Furazolidone is a nitrofurant antibiotic

and a broad-spectrum antibacterial drug that can interfere with bacterial oxidoreductase, thereby inhibiting both Gram-positive and Gram-negative bacteria. Levofloxacin belongs to the class of quinolone antibiotics and also exhibits broad-spectrum antibacterial effects, especially against Gram-negative bacteria. It is commonly used in clinical antibacterial therapy. Omeprazole is a proton pump inhibitor that can inhibit gastric acid secretion by accumulating in the gastric wall after absorption in the small intestine, leading to good therapeutic effects. Although the combination of levofloxacin, furazolidone, and omeprazole can improve the condition of patients to some extent, the overall efficacy is not satisfactory and can cause more adverse reactions [11].

Rabeprazole belongs to a new type of proton pump inhibitor widely used in modern clinical practice. After its application, it can inhibit H⁺/K⁺-ATPase and have good effects in reducing acid secretion and treating *Helicobacter pylori* infection. When rabeprazole is combined with levofloxacin and furazolidone, it can attach to the surface of gastric wall cells, thus lowering the pH value in the stomach and increasing the stability of furazolidone and levofloxacin, maximizing their broad-spectrum antibacterial effects and significantly reducing the degree of *Helicobacter pylori* infection [12]. In addition, the use of rabeprazole can improve the recovery efficiency of gastric mucosal gland function, accelerate the microcirculation speed in the stomach, regulate the levels of gastrin and endothelin in the serum, effectively reduce inflammatory reactions, control the progression of the disease, and significantly improve the prognosis of patients [13].

Serum gastrin is an important hormone in the gastrointestinal tract that mainly originates from G cells in the gastric antrum and proximal duodenal mucosa. It stimulates the secretion of bile and pancreatic juice. After chronic atrophic gastritis occurs, the gastric mucosal glands gradually shrink, leading to a significant decrease in serum gastrin levels. Therefore, measuring the serum gastrin level can assess the severity of the patient's condition. Endothelin can further enhance vascular tension and stabilize the cardiovascular system, increase vascular constriction efficiency, and regulate gastric mucosal blood flow. When the blood vessels constrict, blood flow decreases, leading to increased damage to the gastric mucosal function. This study found that all treatment indicators in the observation group were superior to those in the control group. Therefore, the treatment of chronic atrophic gastritis patients with levofloxacin, furazolidone, and rabeprazole ensures good clinical efficacy and safety [14].

In conclusion, the treatment of chronic atrophic gastritis patients with a triple therapy regimen containing rabeprazole has shown good effectiveness and high safety, and is worthy of promotion.

Conflicts of Interest

The author declares that there is no conflict of interest regarding the publication of this article.

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