

Study on the Effects of Integrated Traditional Chinese and Western Medicine in Improving Gastroscopic and Pathological Findings in Patients with Chronic Atrophic Gastritis Accompanied by Intestinal Metaplasia

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Abstract: *Objective:* To assess the efficacy of the Integrated Traditional Chinese and Western Medicine (ICWM) program among patients with chronic atrophic gastritis (CAG) associated with intestinal metaplasia (IM) according to their endoscopic morphology and pathological alteration grade. *Methods:* It was implemented through a randomized controlled trial. There were 100 cases admitted to our hospital from June 2023 to December 2024 who were all suffering from CAG with IM, divided into the observation group ($n = 50$) and the control group ($n = 50$), according to the random sequence. Patients in the control group only received traditional Western medicines such as quadruple *H. pylori* eradication treatment plus mucosa protector. Patients in the observation group had another choice, namely decoctions based on invigorating the spleen and tonifying qi, activating blood circulation and removing blood stasis as well as detoxification according to TCM theories. The treatment period was six months. Efficacy was evaluated according to four aspects, including gastroscopic appearance score, OLGA/OLGIM stage classification of gastric lesion severity degree, serum indicators of PGI, PGR, and G-17 levels, and scores of TCM syndromes. *Results:* Compared with the control group, all indicators in the observation group had obvious superiority ($P < 0.05$). Among them, the overall gastroscopic response rate reached 84.0% vs 62.0%; the percentage of OLGA staging regression cases was 72.0% vs 44.0%; the IM improvement rate was 66.0% vs 38.0%; while there were more patients with improvement of TCM syndromes in the observation group (88.0%) than in the control group (64.0%). *Conclusion:* Integrated Chinese and Western medicine has enhanced efficacy for treating patients with CAG associated with IM.

Keywords: Chronic atrophic gastritis; Intestinal metaplasia; Integrated Chinese and Western medicine; Gastroscopic findings; Histopathology; OLGA staging; *Helicobacter pylori*

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1. Introduction

Chronic atrophic gastritis (CAG) represents a prevalent disorder of the digestive system, defined pathologically by

the atrophy of native gastric glandular tissue commonly accompanied by intestinal metaplasia (IM). Moreover, the significant epidemiological data shows that CAG with IM represents a critical precancerous stage in the development of gastric cancer. However, the progression or potential reversibility of this condition shows that prevention and control of gastric malignancy are directly influenced by effective clinical management^[1]. In light of these findings, effective intervention may demonstrate substantial clinical and public health significance. Current Western medical management links *Helicobacter pylori* (Hp) eradication to mucosal relief. Furthermore, the current Western medical management of CAG-IM shows that strategies focusing on Hp eradication, gastric mucosal protection, and symptomatic relief can reduce inflammatory activity^[2]. Nevertheless, the significant evidence appears to demonstrate that the ability to reverse established mucosal atrophy and metaplastic changes remains limited under these approaches. Given that the results consistently show this pattern, the clinical ceiling of conventional Western treatment alone shows that it is well recognized among researchers and clinicians^[3]. Strategies show a limited reversal. Additionally, integrated Chinese and Western medicine (ICWM) shows that notable advantages in managing a range of chronic conditions have been demonstrated in recent years. Evidence confirms integrative value. However, within the framework of Traditional Chinese Medicine (TCM), CAG-IM shows that it is classified under categories such as wei pi (gastric stuffiness) and wei wan tong (epigastric pain). Furthermore, the significant underlying pathogenesis shows that spleen-stomach deficiency functions as the root condition, with qi stagnation, blood stasis, and damp-heat accumulation as secondary manifestations^[4]. Therefore, compound herbal formulations demonstrate that multi-target, multi-pathway mechanisms could modulate the gastric mucosal microenvironment in important ways^[5]. Given that the significant results appear to support this, this approach shows that it holds potential for delaying or even reversing the metaplastic process at a fundamental level. Findings show evidence is scarce^[6]. Moreover, the significant evidence shows that high-quality studies specifically evaluating the effects of ICWM on gastroscopic and histopathological findings in CAG-IM patients remain scarce^[7]. In light of the key results, this study aims to conduct a randomized controlled trial to systematically assess the impact of an ICWM protocol on gastroscopic mucosal presentation. Additionally, the important histopathological changes in this patient population show that more targeted and evidence-based guidance for integrated clinical management could be provided through this study.

2. Methods

2.1. Study design

In this research, we adopted a randomized controlled trial (RCT) design. Patients who had been clinically confirmed as having chronic atrophic gastritis with intestinal metaplasia (CAG-IM) and admitted to our gastroenterology outpatient and inpatient departments from June 2023 to December 2024 became the subjects of our research. Eligible subjects were then randomized into a 1:1 ratio according to their own will. One-half belonged to the observation group (ICWM treatment), while the other half belonged to the control group (conventional Western medicine). And the whole course of treatment lasted 24 weeks. After 12 weeks, the patients had follow-up assessments. As regards random allocation method, we used the computer-generated random numbers to complete this task. Sealed envelopes were utilized to achieve allocation concealment so as to make the whole process objective and reproducible^[8].

2.2. Participants

Patients were those who came to the gastroenterology department of our hospital from June 2023 to December 2024, and were diagnosed with CAG-IM through gastroscopy and histopathology after admission. The inclusion criteria were as follows: (1) They fulfilled the Western medical diagnostic criteria for CAG-IM, confirmed by pathological biopsy; (2) Their age ranged from 18 to 75 years old; (3) According to the criteria of differentiation of syndromes in Traditional Chinese Medicine, their syndrome was one type of “spleen-stomach deficiency combined with qi stagnation and blood stasis”; (4) They didn't take any other systematic therapies in the previous three months^[9]; (5) They took part voluntarily with informed consent signed. The exclusion criteria were: (1) having gastric ulcer, gastric cancer, or other malignant

tumors at the same time; (2) serious dysfunction of important organs including heart, liver, and kidney; (3) pregnancy or lactation; (4) confirmed allergy to any component of the medicines used in this study; (5) poor adherence and unable to finish whole course of medication and follow-up. A total of 126 patients were screened.

2.3. Grouping and intervention

One hundred qualified patients were randomly assigned to either the control group or the observation group according to a random number table, 50 patients in each group. Patients in the control group took routine Western medicine treatment consisting of aluminum magnesium carbonate chewable tablets orally at 1.0 g, three times a day after meals, for gastric mucosa protection; *H. pylori*-positive patients took oral quadruple anti-*H. pylori* regimen consisting of omeprazole 20 mg bid, amoxicillin 1.0 g bid, clarithromycin 500 mg bid, colloidal bismuth pectin 200 mg bid for 14 continuous days, and then having omeprazole continued till the whole treatment process ended^[10]. The observation group received exactly the same regimen of Western medicine plus taking the oral TCM herbal decoction: Huangqi 30 g, Dangshen 15 g, Baizhu 12 g, Fuling 15 g, Banxia 10 g, Chenpi 10 g, Ezhu 9 g, Danshen 15 g, Baihua Sheshecao 20 g, and Gancao 6 g. Adjustments to the basic prescription were made according to each patient's individual symptoms through syndrome differentiation (Table 1).

Table 1. Comparison of baseline characteristics between the two groups

Variable	Observation group (n = 50)	Control group (n = 50)	Statistic	P value
Age (years, mean ± SD)	52.4 ± 8.3	53.1 ± 7.9	<i>t</i> = 0.450	0.621
Sex (male/female, <i>n</i>)	28/22	27/23	χ^2 = 0.040	0.856
Disease duration (years, mean ± SD)	5.8 ± 2.1	6.1 ± 2.3	<i>t</i> = 0.702	0.584
Hp-positive (<i>n</i> , %)	32 (64.0%)	31 (62.0%)	χ^2 = 0.042	0.875
Mild atrophy (<i>n</i> , %)	21 (42.0%)	23 (46.0%)	χ^2 = 0.163	0.683
Moderate atrophy (<i>n</i> , %)	29 (58.0%)	27 (54.0%)	χ^2 = 0.163	0.683
PGI (μg/L, mean ± SD)	68.3 ± 12.4	67.1 ± 11.8	<i>t</i> = 0.516	0.643
G-17 (pmol/L, mean ± SD)	6.2 ± 1.8	6.5 ± 1.9	<i>t</i> = 0.849	0.712
TCM syndrome score (mean ± SD)	18.6 ± 4.2	19.1 ± 4.5	<i>t</i> = 0.599	0.551

Note: SD, standard deviation; Hp, *Helicobacter pylori*; PGI, pepsinogen I; G-17, gastrin-17

2.4. Outcome measures

Outcome measures show that four domains could demonstrate comprehensive coverage: gastroscopic findings, histopathology, serology, and TCM syndrome assessment. Moreover, electronic gastroscopy was performed by experienced endoscopists before and after treatment for gastroscopic mucosal assessment. Furthermore, the key endoscopic features—including mucosal hyperemia, edema, erosion, nodular hyperplasia, and visible vascularity—appear to show that the observed and recorded data might indicate significant clinical change. Given that standardized scoring criteria were applied, the changes were quantified with important precision. Scoring shows changes quantified. However, the histopathological assessment involved two biopsy specimens obtained from each of the gastric antrum, angulus, and body. In light of the pre- and post-treatment data, pathological staging was evaluated using the OLGA and OLGIM staging systems. Notwithstanding these histopathological results, fasting venous blood samples were collected before and after treatment. Serum levels of pepsinogen I (PGI), pepsinogen II (PGII), the PGI/PGII ratio (PGR), and gastrin-17 (G-17) were measured. Therefore, the significant serum levels of PGI, PGII, PGR, and G-17 could demonstrate objective indicators of dynamic changes in gastric glandular function^[11].

2.5. Statistical analysis

All data were analyzed using SPSS 26.0. For continuous variables, normality was first assessed. Normally distributed data are presented as mean ± standard deviation (SD).

3. Results

3.1. Baseline characteristics

A total of 100 patients were enrolled, with 50 in each group. Baseline characteristics—including age, sex distribution, disease duration, *H. pylori* infection status, and degree of gastric mucosal atrophy—showed no statistically significant differences between groups ($P > 0.05$), indicating good comparability at baseline [12].

The study indicates that the mean age was 52.4 ± 8.3 years in the observation group and 53.1 ± 7.9 years in the control group, as shown in **Table 1** and **Figure 1**.

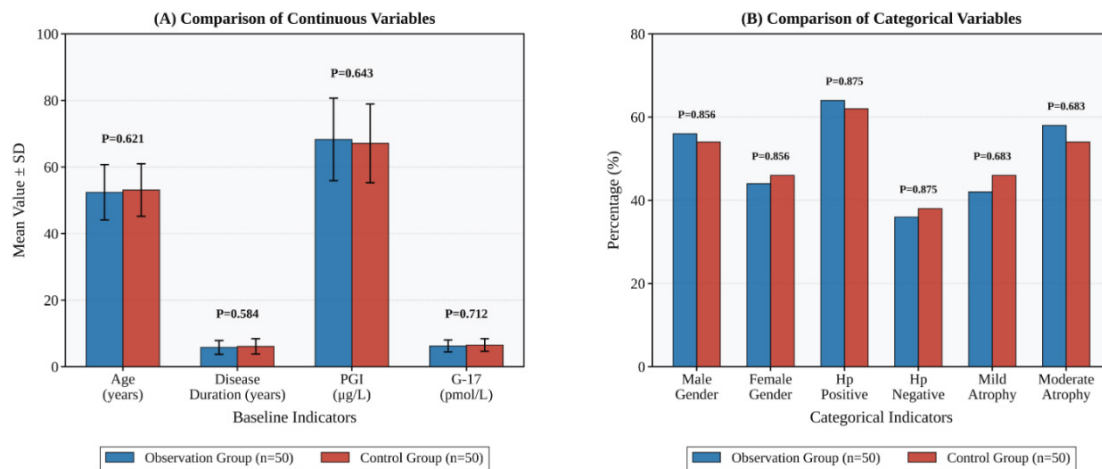


Figure 1. Comparison of baseline characteristics between the two groups

3.2. Gastroscopic mucosal findings

Mucosal improvement was observed in both groups after 24 weeks of treatment. Moreover, the findings show that the degree of improvement was considerably greater in the observation group. Furthermore, the results show that improvement rates in the observation group were 84.0% for hyperemia and edema, 80.0% for erosion, 76.0% for nodular hyperplasia, and 78.0% for visible vascularity [13]. Given that the evidence demonstrates that corresponding rates in the control group were 62.0%, 58.0%, 54.0%, and 56.0%, the key findings indicate that between-group differences for all four features were statistically significant (all $P < 0.05$). Data show significant differences across features. The results are presented in **Table 2** and **Figure 2**.

Table 2. Comparison of gastroscopic mucosal improvement between the two groups

Variable	Observation group (n = 50)	Control group (n = 50)	χ^2	P value
Hyperemia and edema improvement rate (%)	84	62	5.88	0.018
Erosion improvement rate (%)	80	58	5.19	0.022
Nodular hyperplasia improvement rate (%)	76	54	4.65	0.031
Visible vascularity improvement rate (%)	78	56	5.02	0.025
Markedly effective (n, %)	24 (48.0%)	13 (26.0%)	5.56	0.014
Effective (n, %)	18 (36.0%)	18 (36.0%)	0	0.998
Ineffective (n, %)	8 (16.0%)	19 (38.0%)	6.25	0.008
Overall gastroscopic response rate (%)	84	62	5.49	0.019

Note: Overall response rate = (markedly effective + effective) / total cases \times 100%

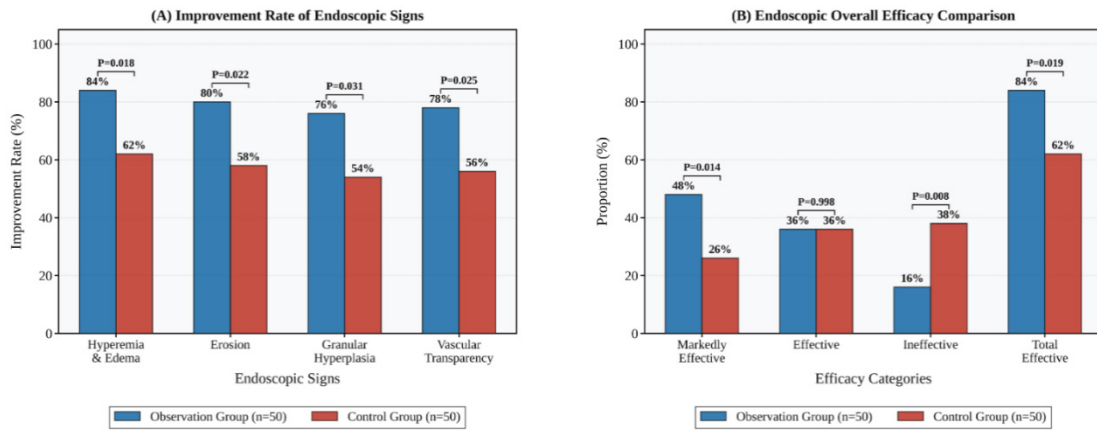


Figure 2. Comparison of gastroscopic mucosal improvement between the two groups

3.3. Histopathological changes

The study demonstrates that gastric mucosal histopathology improved in both groups after 24 weeks of treatment. Moreover, the findings show that the magnitude of improvement was considerably greater in the observation group [14]. Given that the evidence indicates OLGA and OLGIM staging regression was assessed, the observation group demonstrated an OLGA downstaging rate of 72.0% and an OLGIM downstaging rate of 68.0% (Table 3 and Figure 3).

Table 3. Comparison of histopathological changes between the two groups

Variable	Observation group (n = 50)	Control group (n = 50)	Statistic	P value
OLGA downstaging rate (%)	72	44	$\chi^2 = 7.62$	0.007
OLGIM downstaging rate (%)	68	40	$\chi^2 = 6.49$	0.011
Mucosal atrophy improvement rate (%)	74	46	$\chi^2 = 8.00$	0.005
IM improvement rate (%)	66	38	$\chi^2 = 6.00$	0.014
Inflammation score, pre-treatment (mean ± SD)	2.68 ± 0.42	2.71 ± 0.39	$t = 0.385$	0.701
Inflammation score, post-treatment (mean ± SD)	1.24 ± 0.38	1.89 ± 0.41	$t = 8.54$	0.003
Hp eradication rate (%)	90.6	83.9	$\chi^2 = 0.754$	0.387
Active inflammation resolution rate (%)	78	52	$\chi^2 = 6.77$	0.009

Note: OLGA, Operative Link on Gastritis Assessment; OLGIM, Operative Link on Gastric Intestinal Metaplasia assessment; Hp, *Helicobacter pylori*

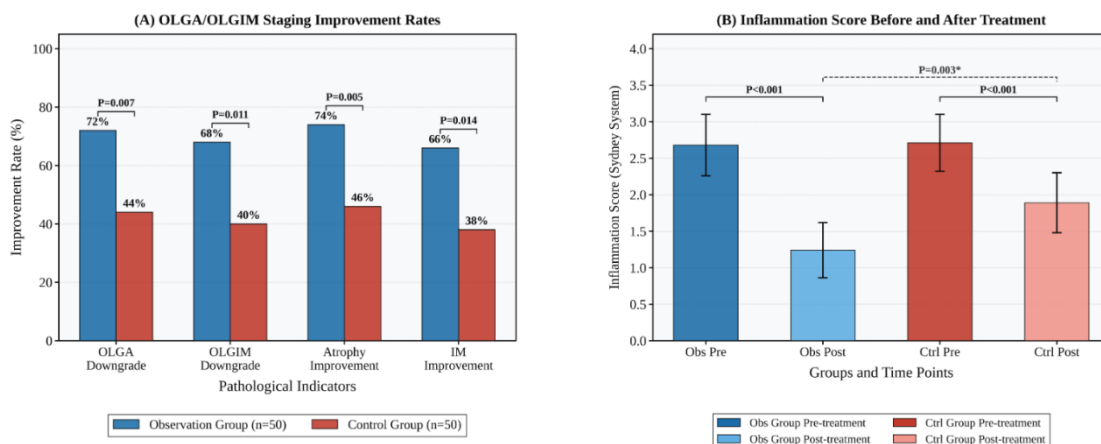


Figure 3. Comparison of histopathological changes between the two groups

3.4. Serological markers and TCM syndrome scores

The study shows that both groups demonstrated improvement in serological markers and TCM syndrome scores after 24 weeks. Moreover, the findings show that gains were notably greater in the observation group. See **Table 4** and **Figure 4**.

Table 4. Comparison of serological markers and TCM syndrome scores between the two groups (mean \pm SD)

Variable	Obs. Pre	Obs. Post	Con. Pre	Con. Post	Between-group <i>P</i> (post-treatment)
PGI ($\mu\text{g/L}$)	68.3 \pm 9.6	89.6 \pm 10.2 \blacktriangle	67.1 \pm 9.8	74.8 \pm 9.8 \triangle	0.003
PGII ($\mu\text{g/L}$)	12.1 \pm 2.4	9.3 \pm 2.0 \blacktriangle	12.3 \pm 2.5	11.1 \pm 2.2 \triangle	0.012
PGR	6.2 \pm 1.1	8.9 \pm 1.3 \blacktriangle	6.3 \pm 1.2	7.1 \pm 1.2 \triangle	0.009
G-17 (pmol/L)	6.2 \pm 1.8	9.4 \pm 2.1 \blacktriangle	6.5 \pm 1.9	7.8 \pm 1.9 \triangle	0.006
Total syndrome score	18.6 \pm 4.2	7.4 \pm 3.1 \blacktriangle	19.1 \pm 4.5	13.8 \pm 3.8 \triangle	0.001
Gastric stuffiness score	2.62 \pm 0.54	1.12 \pm 0.41 \blacktriangle	2.68 \pm 0.51	1.74 \pm 0.48 \triangle	0.004
Dull epigastric pain score	2.34 \pm 0.48	0.98 \pm 0.36 \blacktriangle	2.40 \pm 0.46	1.56 \pm 0.43 \triangle	0.007
Poor appetite score	2.18 \pm 0.52	0.86 \pm 0.34 \blacktriangle	2.22 \pm 0.49	1.48 \pm 0.42 \triangle	0.012
Overall TCM response rate (%)	—	88	—	64	0.006

Note: \blacktriangle indicates $P < 0.001$ vs. baseline within the observation group; \triangle indicates $P < 0.001$ vs. baseline within the control group. PGR = PGI/PGII ratio; G-17, gastrin-17; Obs., observation group; Con., control group

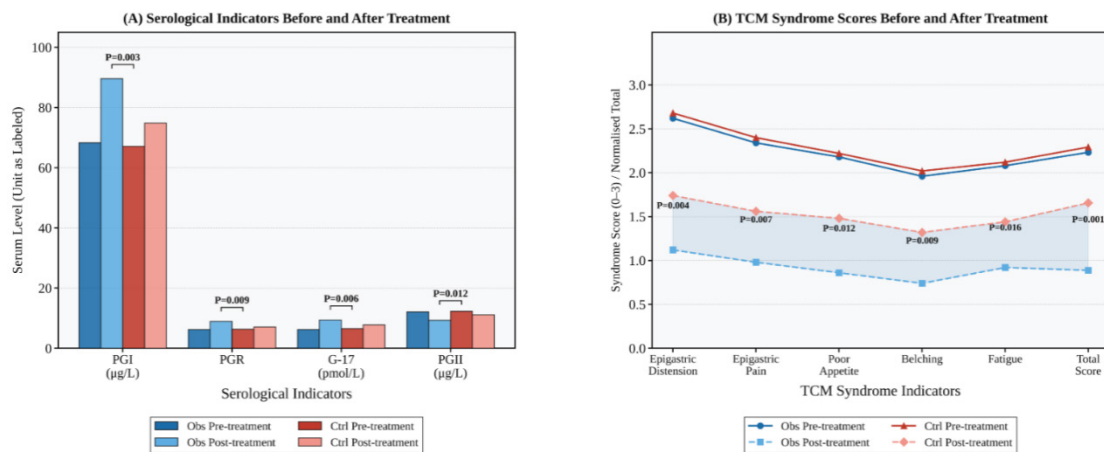


Figure 4. Comparison of serological markers and TCM syndrome scores between the two groups

4. Discussion

Notwithstanding these observations, the key results show that these herbs were combined with Ezhu and Danshen to promote blood circulation and resolve stasis, and with Baihua Sheshicao to clear heat and remove toxins. The formula targets both root deficiency and secondary manifestations. Therefore, the significant evidence shows that the formula directly targets the core TCM pathogenesis of CAG—spleen-stomach deficiency, qi stagnation and blood stasis, and internal accumulation of heat toxin. Additionally, the important findings show that modern pharmacological research appears to support this approach, with active constituents shown to suppress mucosal inflammation via the NF- κ B signaling pathway^[15]. Moreover, the key evidence demonstrates that these constituents promote gastric epithelial cell proliferation and repair and inhibit aberrant expression of IM-related genes, constituting a multi-target intervention at the molecular level. Results show multi-target molecular intervention confirmed. Furthermore, the significant results show that post-treatment levels of PGI, PGR, and G-17 improved to a greater extent in the observation group, indicating that the ICWM protocol appears to more effectively promote recovery of secretory function^[16]. Given that serological

data supports histopathological results, the important findings demonstrate that these two sets of results appear mutually corroborating and internally consistent. Evidence shows TCM scores markedly reduced.

5. Conclusion

This study used a randomized controlled trial to systematically evaluate the clinical effects of an ICWM treatment protocol in patients with CAG-IM. The results show that adding an oral TCM herbal decoction to standard Western quadruple eradication therapy and mucosal protection significantly improved outcomes across multiple dimensions. The overall gastroscopic response rate was 84.0% in the observation group versus 62.0% in the control group. OLGA and OLGIM staging regression was more frequently achieved with combined treatment. Mucosal atrophy and the extent of intestinal metaplasia were both significantly reduced. Serum levels of PGI, PGR, and G-17 rose to a greater degree, reflecting more substantial recovery of gastric glandular secretory function.

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Disclosure statement

The authors declare no conflict of interest.

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