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Progress in the Treatment of Chronic Atrophic Gastritis and Precancerous Gastric Lesions in Traditional Chinese and Western Medicine

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Abstract: *Chronic atrophic gastritis (CAG), a prevalent gastric mucosal lesion, is one of the most common diseases of the digestive system. Its clinical management faces dual challenges of low cure rates and high recurrence risks, with a significant potential for progression to malignant lesions, posing a serious threat to patients' health. Notably, CAG has a complex, multifactorial pathogenesis that remains incompletely understood. Current clinical intervention strategies include traditional Chinese medicine (TCM), modern Western medicine, and integrative approaches combining both, demonstrating synergistic advantages in improving clinical symptoms. This article systematically reviews recent domestic studies and discusses the following dimensions: (1) Exploration of etiology and pathogenesis: elucidating core pathogenesis concepts such as "deficiency, stasis, and toxicity"; (2) Therapeutic methods and prescriptions: summarizing characteristic TCM approaches like strengthening the spleen and replenishing Qi, promoting blood circulation and removing stasis, as well as detoxifying and resolving masses; (3) Advances in therapeutic techniques: consolidating clinical research findings on TCM therapies including herbal formulas, acupuncture, and acupoint application; 4) Progress in Western medical diagnostic and treatment technologies. Through a multidimensional analysis, this work aims to provide theoretical foundations and practical insights for the prevention and treatment of this condition.*

Keywords: *Chronic Atrophic Gastritis; Precancerous Gastric Lesions; Traditional Chinese Medicine Diagnosis and Treatment; Western Medicine Therapy*

1. Introduction

Chronic atrophic gastritis (CAG) refers to a chronic gastric disease characterized by the repeated damage of gastric mucosal epithelium, leading to a reduction in intrinsic glands, which may be accompanied by intestinal metaplasia and/or pseudopyloric metaplasia [1]. This condition shows significant age-related prevalence, with epidemiological studies indicating an increasing incidence with advancing age. According to the gastric cancer progression model proposed by Correa, the sequence follows: normal gastric mucosa → superficial gastritis → chronic atrophic gastritis (CAG) → intestinal metaplasia → dysplasia → gastric cancer [2]. Dysplasia is identified as a direct precancerous lesion, with CAG, intestinal metaplasia, and dysplasia collectively categorized as precancerous lesions of gastric cancer (PLGC) [3]. Therefore, early diagnosis and treatment of CAG and PLGC are critical to reducing the incidence of gastric cancer. Clinical manifestations of CAG are non-specific, with many patients being asymptomatic. Others may present with non-specific dyspeptic symptoms such as upper abdominal discomfort, bloating, fullness, or pain, accompanied by digestive symptoms such as poor appetite, heartburn, belching, acid reflux, nausea, or a bitter taste in the mouth. Additionally, systemic and psychological symptoms like fatigue, weight loss, forgetfulness, anxiety, or depression can occur, often triggered by factors such as improper diet, poor emotional state, imbalanced work-rest schedules, or changes in weather [1,4].

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2. Advances in Traditional Chinese Medicine (TCM) Treatment

2.1 Etiology and Pathogenesis

In Traditional Chinese Medicine (TCM), there are no specific terms for chronic atrophic gastritis (CAG) and precancerous lesions of gastric cancer (PLGC). However, based on symptoms such as upper abdominal fullness, pain, and distention, these conditions are classified under "Weipi" (gastric obstruction) and "Weiwantong" (gastric pain). "Weipi" was first recorded in Zhang Zhongjing's Treatise on Cold Damage, which describes fullness without pain as "Pi," while The Spiritual Pivot: Meridians introduced "Weiwantong" along with its associated symptoms: gastric pain, distention, belching, and relief following exhalation. TCM identifies the stomach, spleen, and liver as the primary affected organs, with a pathological basis of spleen and stomach weakness and Qi deficiency. Factors such as irregular diet, external pathogens, emotional disturbances, or medication-induced damage disrupt transportation and transformation processes, leading to the production of pathological byproducts like dampness, phlegm, and stasis. Professor Yao Naili summarized the pathogenesis as "liver stagnation, spleen deficiency, network obstruction, and toxic damage," rooted in spleen Qi and stomach Yin deficiency, evolving into liver stagnation, blood stasis, heat, and internal toxin generation. Professor You Shaowei described CAG as a mixture of deficiency and excess, marked by cold and heat, with spleen deficiency as the foundation and stagnation as the critical condition for disease progression. Professor Huang Bin described CAG as mixed deficiency and excess, with "deficiency" including spleen Qi and stomach Yin insufficiency, and "excess" characterized by Qi stagnation, liver depression, blood stasis, damp-heat, and turbid toxins. Professor Wang Xixing highlighted CAG's complex etiology, with early stages dominated by mixed cold and heat, mid-stages by liver-stomach disharmony, and late stages by Qi deficiency and blood stasis.

TCM believes that the primary locations of these diseases are in the stomach, spleen, and liver. The pathological basis is spleen and stomach deficiency with insufficient vital energy. The main causes include improper diet, external pathogenic factors, emotional disturbances, irregular work and rest, or drug-induced injury, which ultimately injure the middle burner, disrupt normal transformation and transportation, and lead to disordered qi movement and the production of pathological products such as "dampness, phlegm, and blood stasis." Professor Yao Naili [5] summarized the basic pathogenesis of CAG as "liver depression and spleen deficiency, with network obstruction and toxin damage," considering spleen qi deficiency and stomach yin deficiency as the root, followed by liver depression on the basis of spleen deficiency, blood stasis due to long-term illness, heat from stasis, and the internal generation of heat toxins. You Shaowei [6] and others believe that CAG often presents with a mixture of deficiency and excess, with both cold and heat symptoms, and is characterized by spleen deficiency and stagnation. Spleen deficiency is the fundamental cause, stagnation is an important condition for the occurrence of CAG, and stasis is the key. Professor Huang Bin [7] believes that CAG is a condition of mixed deficiency and excess, with "deficiency" being spleen qi deficiency and stomach yin deficiency, and "excess" being qi stagnation, liver depression, blood stasis, damp-heat, and turbid toxins. Professor Wang Xixing[8] believes that the etiology and pathogenesis of CAG are complex, with initial stages dominated by a mixture of cold and heat, middle stages by disharmony between the liver and stomach, and late stages by qi deficiency and blood stasis.

2.2 Syndrome Differentiation and Treatment

The Expert Consensus on Integrated Traditional Chinese and Western Medicine for Chronic Atrophic Gastritis (2017) [1] summarized CAG into six syndromes and provided corresponding prescriptions: for liver-stomach qi stagnation syndrome, use Bupleurum Liver-Regulating Powder with modifications; for liver-stomach depression-heat syndrome, use Modified Bupleurum and Scutellaria Decoction combined with Zuo Jin Wan with modifications; for spleen-stomach deficiency syndrome (spleen-stomach yang deficiency syndrome), use Astragalus Stomach-Building Decoction with modifications; for spleen-stomach damp-heat syndrome, use Modified Lianpu Drink with modifications; for stomach yin deficiency syndrome, use Modified Yiguan Decoction combined with Peony and Licorice Decoction with modifications; for stomach network blood stasis syndrome, use Modified Shixiao Powder combined with Danshen Drink with modifications. Master of Traditional Chinese Medicine Wang Qingguo [9] divided CAG into three syndromes: spleen-stomach qi deficiency syndrome, spleen-stomach damp-heat syndrome, and blood stasis in the stomach network syndrome. Based on his clinical experience, he treated patients with spleen-stomach qi deficiency syndrome using his self-prescribed Gui Pi Jian Zhong Anti-Atrophy Decoction, patients with spleen-stomach damp-heat syndrome using Modified Baiwu Xie Xin

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Tang, and patients with blood stasis in the stomach network syndrome using a combination of spleen-supplementing and qi-benefiting herbs with blood-activating and stasis-eliminating ingredients. Professor Li Jing [10] divided CAG into three syndromes: treating spleen-stomach qi deficiency syndrome with modified Jianpi Tang, treating spleen-stomach yin deficiency syndrome with modified Taizi Shen Wumei Fang, and treating liver depression and spleen deficiency syndrome with modified Sini San or Bupleurum Liver-Regulating Powder combined with Liu Junzi Tang. Professor Zhang Jize [11] divided CAG into four major syndromes: central deficiency and stomach cold syndrome, liver-stomach disharmony syndrome, stomach yin deficiency syndrome, and qi stagnation and blood stasis syndrome, and treated them with modified Xiao Jian Zhong Tang, Bupleurum Liver-Regulating Powder, Shashen Maidong Tang, and Xuefu Zhuyu Tang, respectively. Huang Yuping et al. [12] analyzed the symptoms of 110 patients with PLGC and divided them into six syndromes: spleen-stomach deficiency, spleen-stomach damp-heat, liver-stomach depression-heat, liver-stomach qi stagnation, stomach yin deficiency, and stomach network blood stasis.

2.3 Treatment with Empirical Prescriptions

Zeng Weiwei et al. [13] found that Liqi Tongluo Yuwei Decoction exerts anti-inflammatory effects by inhibiting the release of inflammatory mediators, offering multi-target protection for gastric mucosal glands and their functions. Zuo Qiaoyun et al. [14] demonstrated that Qizhu Jianwei Decoction improves gastric function, promotes mucosal repair, and inhibits the expression of proteins such as Lgr5, Wnt1, and C-myc, thereby blocking or reversing the progression of CAG and PLGC. Li Jing [15] highlighted that the core pathogenesis of CAG lies in spleen-stomach Qi deficiency, which leads to blood stasis and impaired nourishment of the spleen and stomach, further exacerbating their dysfunction. Based on Zhizhu Pill, she developed Qizhu Weiwei Decoction, which restores the driving force of Qi circulation, eliminates pathological blood stasis, and improves spleen-stomach malnourishment. This personalized formula significantly alleviates patient symptoms. Fei Baoying et al. [16] discovered that Yiwei Decoction regulates the expression of Vcan, miR-542-3p, lnc32332 in gastric tissues, and miR-363-3p in serum, while also inhibiting the expression of cancer treatment targets VEGFR, HER-2, and EGFR to varying degrees, thereby alleviating the progression of CAG and PLGC. Huang Xiangming et al. [17] demonstrated that Qiling Decoction acts through the PI3K/Akt pathway to regulate gastric intestinal metaplasia (GIM) while reducing serum levels of IL-6, IL-1β, and TNF-α in GIM rats, thereby suppressing inflammatory responses and delaying or partially reversing CAG and PLGC progression. Ren Xiaojun et al. [18] found that Banxia Xiexin Decoction improves gastric mucosal atrophy through multiple mechanisms, aids in eradicating Helicobacter pylori, enhances the gastric microenvironment, and regulates gastrointestinal hormones, thus delaying or reversing CAG and PLGC. Yang Chuqi et al. [19] reported that modified Chaihu Shugan Powder inhibits tumors, repairs gastric mucosa, and enhances immunity, showing efficacy in improving clinical symptoms and delaying or reversing CAG and PLGC. Zhang Dan et al. [20] demonstrated the effectiveness of Huatan Xiaoyu Formula in treating PLGC by alleviating symptoms such as gastric pain and reducing tumor-associated markers, thereby improving intestinal metaplasia reversal rates. Kou Yuan et al. [21] revealed that Yangwei Qingre Huayu Decoction effectively alleviates clinical symptoms and reduces pathological scores of gastric mucosa in patients with CAG and PLGC.

2.4 Acupuncture and Other Therapies

Ji Zhuangzhuang et al. [22] summarized Professor Shao Zuyan's treatment of PLGC patients, incorporating TCM external therapies such as herbal decoctions, herbal plasters, and acupuncture based on syndrome differentiation and constitutional identification, achieving notable clinical efficacy. Zheng Xue et al. [23] found that medicinal cake moxibustion at Zhongwan and Qihai acupoints activates the PTEN-AKT tumor suppressor pathway, affecting the biological functions of MDM2 and Caspase-9, repairing damaged gastric mucosa, and improving pathological gastric mucosal conditions in CAG rats, thus preventing CAG and PLGC. Rong Hua et al. [24] reported that Danshi Jianpi Decoction combined with acupuncture significantly alleviates CAG and PLGC symptoms by modulating the Wnt3/β-catenin signaling pathway, improving clinical signs, delaying gastric fibrosis progression, and relieving TCM symptoms. Zhu Jingru et al. [25] employed Five Element Music Therapy and Emotion-Adjustment Methods to regulate mood, harmonize Qi circulation, improve symptoms, and prevent PLGC progression,achieving remarkable efficacy. Based on the "One-Qi Circulation" theory, Shui Diankui et al. [26] conducted a clinical trial combining Lizhong Tongluo Huazhuo Decoction and acupoint embedding therapy for CAG and PLGC, showing significant symptom improvement and effective reversal of pathological gastric mucosa states.Wang Xiangyang [27] observed that Weishu Mixture combined with acupuncture effectively improves gastrointestinal hormone levels and motility in treating

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spleen-stomach damp-heat type CAG, relieving inflammation and associated symptoms. Wu Can et al. [28] demonstrated that acupuncture combined with modified Xiangsha Liujunzi Decoction has multi-target therapeutic effects for CAG, modulating gastrointestinal motility and enzyme-hormone levels. This integrated therapy not only alleviates symptoms but also improves abnormal gastric mucosal histology, such as glandular atrophy and dysplasia, ultimately achieving significant clinical outcomes.

3. Advances in Western Medicine Treatment

3.1 Etiology and Pathogenesis

Western medicine identifies chronic atrophic gastritis (CAG) and precancerous lesions of gastric cancer (PLGC) as conditions resulting from multiple factors, with Helicobacter pylori (Hp) infection being considered the primary cause. This pathogen carries the cytotoxic cagA gene, which expresses specific carcinogenic proteins. Using its type IV secretion system, Hp injects these proteins directly into gastric mucosal epithelial cells, leading to mucosal damage and, ultimately, glandular atrophy in the stomach [29]. Other contributing factors include bile reflux, damage caused by nonsteroidal anti-inflammatory drugs (NSAIDs), autoimmune disorders, and unhealthy dietary habits.

3.2 Diagnosis of the Disease

Various methods are employed in the diagnosis of CAG and PLGC, including endoscopic examination, gastric mucosal histological analysis, and laboratory tests. However, diagnosis primarily relies on endoscopy and histological examination of gastric mucosa, with the latter offering greater diagnostic value. The Expert Consensus on the Management of Precancerous Gastric Mucosal Conditions and Lesions in China (2020) [30] highlights that, compared to conventional white-light endoscopy, high-definition chromoendoscopy provides more precise guidance for biopsy sampling, delivering high-accuracy tissue specimens and significantly improving the early pathological diagnosis of gastric precancerous lesions. Characteristic endoscopic features in CAG patients include mucosa with alternating red and white coloration, predominantly white, with flattened or absent mucosal folds, and visible submucosal blood vessels. In some cases, the mucosa may appear rough with fine granular or nodular proliferation. Gastric mucosal histological analysis via biopsy demonstrates glandular atrophy or intestinal metaplasia, confirming the diagnosis of CAG [1]. Laboratory tests can complement the diagnostic process, particularly by measuring serum pepsinogen I and II levels and gastrin-17, which help assess the presence and location of gastric mucosal atrophy. Given that Hp infection is a key causative factor in CAG and PLGC, accurate diagnosis of its presence is crucial. Clinically, a urea breath test combined with multi-site biopsies of the gastric antrum and body is often used for precise Hp infection detection, facilitating the diagnosis of CAG and PLGC and enabling early gastric cancer screening.

3.3 Western Medicine Treatment

3.3.1 Pharmacological Treatment

Western medicine primarily utilizes pharmacological approaches to treat chronic atrophic gastritis (CAG), including Helicobacter pylori (Hp) eradication, gastric mucosal protective agents, drugs to improve gastric motility, and antioxidants. Studies [31] have found that Hp eradication can enhance red blood cell immune function, reduce gastric mucosal inflammation, strengthen innate immunity, and regulate specific immunity, thereby promoting rapid recovery. This method is considered effective in CAG treatment. Gastric mucosal damage is an important pathological change inducing CAG. Clinical administration of gastric mucosal protective agents can reduce damage, enhance mucosal resistance, and effectively protect the gastric mucosa [32]. For example, oral colloidal bismuth pectin is commonly used to protect gastric mucosa and relieve clinical symptoms.Drugs that improve gastric motility effectively reduce bile acid reflux, thereby minimizing gastric acid erosion of the mucosa and promoting functional recovery. Modern pharmacological research [33] shows that mosapride, a drug to enhance gastric motility, stimulates gastrointestinal peristalsis, facilitating the restoration of gastric function. Antioxidant therapy has shown clinical efficacy, with folic acid combined with vitamin B12 proven to promote the repair and regeneration of epithelial cells in the digestive tract, leading to significant therapeutic results.

3.3.2 Endoscopic Treatment

With advancements in modern medical technology, endoscopic techniques have become widely

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applied in disease management, transitioning from solely diagnostic tools to vital therapeutic modalities. In the clinical treatment of CAG, endoscopic interventions demonstrate notable advantages, primarily including techniques such as Endoscopic Mucosal Resection (EMR), Endoscopic Submucosal Dissection (ESD), and Argon Plasma Coagulation (APC) [34]. These minimally invasive procedures enable precise resection and separation of pathological and atrophic tissues while simultaneously enhancing diagnostic accuracy and achieving therapeutic goals. For instance, Argon Plasma Coagulation utilizes high-frequency currents generated by ionized argon gas to accurately ablate lesion areas. Its unique hemostatic properties effectively control intraoperative bleeding while promoting coagulation and repair of wound tissue. This dual mechanism ensures treatment safety and operational control, offering innovative solutions for managing gastrointestinal mucosal lesions.

4. Integrated Chinese and Western Medicine Treatment

Both Chinese medicine and Western medicine offer distinct advantages in the treatment of chronic atrophic gastritis (CAG) and precancerous lesions of gastric cancer (PLGC). Clinical practices have proposed integrating Chinese and Western medicine to harness complementary strengths, thereby improving treatment efficacy to a certain extent. Hu Jingyan [35] demonstrated that combining the Chinese patent medicine Weifuchun with a standard quadruple therapy (comprising proton pump inhibitor pantoprazole, gastric mucosal protectant potassium citrate bismuth, and antibiotics clarithromycin and amoxicillin) for CAG patients produces synergistic effects. This combination effectively improves gastrointestinal hormone levels, regulates immune function, and promotes gastric mucosal repair and regeneration. The study confirmed that this integrated approach not only enhances Hp eradication rates but also, through multi-target intervention, significantly delays or even reverses pathological progression of CAG, providing a new optimization pathway for clinical treatment. Similarly, Lv Anding et al. [36] found that combining Banxia Xiexin Decoction with quadruple therapy for CAG and PLGC patients not only eradicates Hp but also modulates gastrin and pepsinogen secretion, thereby alleviating clinical symptoms. Xu Yapei et al. [37] showed in a clinical study that employing Yiyi Huazhu Jiedu Formula combined with Viadestin tablets for CAG and PLGC patients improves disease outcomes through multi-pathway synergistic effects, enhances quality of life, and achieves more significant clinical efficacy.

5. Summary and Prospects

Chinese and Western medicine each has unique advantages in treating CAG and PLGC. Western medicine focuses on eradicating root causes and precise interventions, while Chinese medicine emphasizes syndrome differentiation, holistic regulation, and pathological reversal. In recent years, integrated Chinese-Western treatment models have demonstrated unique benefits in symptom improvement, disease progression delay, and pathological reversal for patients with CAG and PLGC. This combination therapy achieves synergistic effects by leveraging Western medicine's targeted approach to control causative factors and Chinese medicine's overall regulation of the internal environment, thereby delaying atrophic progression and reversing pathology. Research suggests that blood-activating and stasis-removing Chinese medicines (such as Curcuma and Panax Notoginseng) may inhibit intestinal metaplasia and dysplasia. Additionally, Chinese medicine reduces recurrence rates by enhancing spleen and stomach functions and lowering Hp reinfection and symptom recurrence rates.

In conclusion, integrated Chinese-Western diagnostic and treatment models for CAG and PLGC demonstrate complementary characteristics. Chinese medicine, guided by syndrome differentiation, achieves individualized and precise interventions through the flexibility of Chinese herbal compound formulations and dynamic adjustments, especially excelling in alleviating subjective symptoms such as gastric fullness and reduced appetite. However, its clinical application still faces challenges related to standardization within evidence-based medical frameworks. Current clinical research is often limited to single-center or small-sample cohorts, resulting in lower evidence levels. Additionally, the molecular mechanisms of active components in Chinese herbal formulas in repairing gastric mucosa, regulating immune homeostasis, and addressing key pathological processes remain inadequately understood. Synergistic regulatory networks involving pathways such as Wnt/β-catenin and NF-κB require further elucidation through network pharmacology, organoid models, and other advanced technologies.

Future research should adopt multi-modal strategies integrating genomics, metabolomics, and other multidimensional data to construct "disease-syndrome-effect" association models. This would provide scientific support for the modernization of Chinese medicine in treating CAG and PLGC and offer more promising diagnostic and therapeutic strategies to achieve "early prevention and early treatment" of

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gastric cancer.

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