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Research Advances in Traditional Chinese Medicine for the Prevention and Treatment of Chronic Atrophic Gastritis

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Abstract: ***Chronic atrophic gastritis (CAG) is a common and recurrent digestive system disorder characterized by reduced gastric mucosal glands, thinning of the mucosa, and varying degrees of intestinal metaplasia or dysplasia. This condition is considered a critical transitional stage from normal gastric mucosa to gastric cancer, making its early prevention and treatment crucial for interrupting the inflammation-to-cancer progression. In recent years, traditional Chinese medicine (TCM) has demonstrated unique advantages in preventing and treating CAG. It not only effectively alleviates clinical symptoms and slows disease progression but also achieves pathological reversal of gastric mucosal changes to some extent, thereby improving patients' quality of life. This paper systematically reviews domestic and international research on TCM treatment for CAG over the past five years. It examines etiology and pathogenesis, syndrome differentiation and classification, therapeutic principles and methods, as well as various treatment modalities including herbal formulas and acupuncture. The study summarizes clinical efficacy and delves into the underlying mechanisms, including anti-inflammatory effects, immune regulation, mucosal repair promotion, and signaling pathway modulation. The aim is to provide theoretical foundations and clinical references for the integrated prevention and treatment of CAG through both TCM and Western medicine.***

Keywords: Chronic Atrophic Gastritis, Traditional Chinese Medicine Syndrome Differentiation and Treatment, Mechanism Research, Research Progress.

1. Introduction

Chronic atrophic gastritis (CAG) is a chronic inflammatory disease characterized by atrophy of the gastric mucosal glands. Clinical manifestations are often nonspecific, commonly including upper abdominal pain, decreased appetite, belching, acid reflux, and nausea. The WHO classifies it as a precancerous condition, with mucosal intestinal metaplasia and dysplasia considered precancerous lesions. Disease progression typically follows the sequence: non-atrophic gastritis → atrophic gastritis → intestinal metaplasia → carcinoma in situ → gastric cancer [1]. The overarching principles of modern medical treatment for CAG focus on symptom relief, reducing gastric mucosal inflammation, preventing bile reflux, supplementing vitamins, protecting the gastric mucosa, and delaying carcinogenesis. Currently, there are no specific drugs capable of reversing or blocking the "inflammation-to-cancer transformation" process in CAG. In recent years, traditional Chinese medicine (TCM), leveraging its multi-targeted and multi-pathway characteristics, has made significant contributions to delaying, blocking, or even reversing the "inflammation-to-cancer transformation" process in CAG [2,3]. This approach, grounded in holistic principles and syndrome differentiation, provides new theoretical foundations and innovative prevention strategies for clinical treatment.

2. Pathogenesis

Traditional Chinese medicine classifies this condition under the categories of "stomach fullness" and "stomach pain," with the earliest documentation found in the Ling Shu section of the Huangdi Neijing [4]: "Those afflicted with stomach disorders experience abdominal distension,with pain centered in the epigastric region." Regarding the pathogenesis, the Suwen of the Neijing states: "Cold pathogens invade the space between the stomach and intestines, beneath the peritoneum, causing blood stagnation and constriction of the minor vessels, thus inducing pain." This emphasizes that external cold pathogens attacking the gastrointestinal tract lead to stagnation of qi and blood, where obstruction causes pain. Li Dongyuan [5], the founder of the Earth-Supplementing School, repeatedly noted in his works that internal injury from improper diet is the primary factor damaging the spleen and stomach. For instance, in Secret Treasures of the Orchid Chamber: Abdominal Distension and Fullness, it states: "Those who consume excessive cold foods or suffer from prolonged spleen-stomach deficiency may experience distension and fullness due to cold in the stomach." Similarly, in Discourse on the Spleen and Stomach: On the Prosperity and Decline of the Spleen and Stomach, it records: "Irregular eating habits lead to stomach disorders; stomach disorders cause shortness of breath and diminished vitality... When the stomach is diseased, the spleen receives no nourishment and thus also becomes diseased." Integrating the theories of the Inner Canon with clinical practice, Master Dongyuan proposed the academic principle that "internal injury to the spleen and stomach gives rise to a hundred diseases," thereby establishing his distinctive theory of internal injury to the spleen and stomach. The liver governs the free flow of qi and regulates its movement, maintaining a close relationship with the spleen and stomach. Emotional imbalance is also a significant factor in the onset of this condition. As stated in Jingyue Quanshu [6]: Fullness and Distension: "Sudden anger violently injures the liver; liver qi remains unresolved, thus causing fullness." Among the seven emotions, particular emphasis is placed on the impact of excessive thinking on the spleen and stomach. The Ling Shu:

The Essence of the Spirit records: “Thoughts originate from the heart, and the spleen responds to them.” In summary, descriptions from ancient medical authorities reveal that the pathogenesis of fullness and stomach pain primarily stems from external pathogens invading the body, dietary imbalances damaging the stomach, and emotional stagnation. These factors collectively lead to spleen-stomach deficiency and disrupted ascending-descending functions.

With advances in medical technology, the integration of macro-level TCM syndrome differentiation with micro-level disease diagnosis via digestive endoscopy has enabled modern practitioners to gain a more comprehensive understanding of the pathogenesis of this condition. Zhang Xichun, a prominent figure in modern integrated Chinese and Western medicine, stated: “The normal function of Yangming stomach qi is its continuous downward movement.” If stomach qi fails to descend and qi mechanism reverses upward, it leads to epigastric and chest fullness and oppression. Concurrently, additional symptoms such as “constipation,” “vomiting,” “hiccups,” and ‘epistaxis’ may manifest [7]. Professor Yang Shaohua emphasized the liver-stomach relationship in clinical practice, stressing that “in relation to the spleen and stomach, the liver is both the organ initiating movement and the organ concluding it.” “Excess qi manifests as heat.” In the early stages of disease, stagnation of liver and stomach qi gradually transforms into heat. When liver-stomach heat accumulates without resolution, it causes burning pain or discomfort in the epigastrium, restlessness, and irritability. Gastroscopy may reveal acute active inflammatory reactions in the gastric mucosa, accelerated gastric motility, and possible bile reflux [8]. Professor Li Dangui, a Master of Traditional Chinese Medicine, posits that the pathogenesis of this disease stems from turbid toxins obstructing qi movement. Prolonged qi stagnation leads to blood stasis in the gastric collaterals, causing the spleen and stomach to lose their nourishing function [9]. Professor Tian Yaozhou posits that in the late stages, stagnation, dampness, blood stasis, and deficiency accumulate over time to form toxins. Spleen deficiency leads to toxin accumulation, which damages gastric collaterals, ultimately causing pathological changes such as intestinal metaplasia, dysplasia, and even carcinogenesis. The Yin Fire theory is one of the core tenets of Li Dongyuan’s Spleen-Stomach theory [10]. Tang Xudong et al., through exploring the “Yin Fire Theory,” discovered its close relationship with the progression of chronic atrophic gastritis with intestinal metaplasia. Spleen-stomach qi deficiency and insufficiency of original qi allow yin fire to arise from the kidneys, stimulating pathological hyperactivity of the heart’s sovereign fire. This fire descends through the meridians to the small intestine, scorching the epigastrium and causing glandular atrophy. Moreover, the fire of the small intestine, following the nature of fire to ascend, triggers the appearance of intestinal epithelial cells—which should only be present in the intestines—within the gastric cavity [11].

3. Differential Diagnosis and Treatment

3.1 Dialectical Classification

Consensus on Integrated Traditional Chinese and Western Medicine Diagnosis and Treatment of Chronic Atrophic Gastritis (2017) [12] categorizes CAG into six patterns: Liver-Stomach Qi Stagnation Pattern (Chaihu Shugan San), Liver-Stomach Heat-Stagnation Pattern (Hualan Decoction combined with Zuojin Pill), Spleen-Stomach Deficiency Pattern (Huangqi Jianzhong Decoction), Spleen-Stomach Damp-Heat Pattern (Lianpu Decoction), Stomach Yin Deficiency Pattern (Yiguan Decoction combined with Shaoyao Gancao Decoction), and and Stomach Collateral Blood Stasis Pattern (Shi Xiao San combined with Danshen Decoction with modifications). CAG represents a pattern of underlying deficiency with superficial excess, or a mixture of deficiency and excess. Clinically, these patterns may present individually or in combination, and different practitioners hold varying perspectives and experiences regarding treatment. Through systematic statistical analysis, Zhang Weijian et al. [13]. investigated the pattern elements, syndrome patterns, and medication patterns used by Master Traditional Chinese Medicine practitioners in treating CAG. They concluded that spleen-stomach qi deficiency, liver-stomach disharmony, and qi stagnation with blood stasis permeate the entire disease progression. They particularly emphasized that spleen-stomach qi deficiency is the primary syndrome, and blood stasis in the stomach collaterals as the primary syndrome leading to the progression of gastric mucosal atrophy to malignancy. Therefore, treatment should focus on strengthening the spleen and harmonizing the stomach, supplemented by methods to regulate qi, activate blood circulation, transform dampness, and eliminate turbidity based on the specific manifestations of qi stagnation, blood stasis, or damp obstruction. Professor Wang Qingguo [14] employs phase differentiation to delineate the disease mechanism into three stages: “Spleen-Stomach Qi Deficiency → Spleen-Stomach Damp-Heat → Stagnation Obstructing the Gastric Collaterals.” For this progression, he proposes distinct primary treatment approaches at each stage: tonifying the spleen and invigorating qi, clearing heat and draining dampness, and activating blood circulation to resolve stasis. Formulas such as Gui Pi Jian Zhong Kang Wei Tang, Bai Wu Xie Xin Tang, and herbs like Salvia miltiorrhiza, Acorus tatarinowii, Curcuma zedoaria, and Corydalis yanhusuo are selected for timely intervention to halt disease progression. Professor Ansheng’s approach to treating elderly CAG patients begins with the theory of “perpetual yin deficiency [15],” proposing the principle that “removing pathogenic factors stabilizes the stomach, while restoring yin nourishes it.” Clinically, he employs Yiguan Decoction augmented with yin-nourishing herbs like white peony root and dendrobium to nourish yin, soothe the liver, and dispel heat. Additionally, he uses Lianpu Decoction with reduced auxiliary doses of dendrobium and Lugen to nourish yin, transform dampness, and clear heat. His proprietary Yigwei Huayu Decoction (Dendrobium 15g, Ophiopogon 10g, Dioscorea 15g, Atractylodes 15g, Bletilla 10g, Salvia 15g, Paeonia rubra 15g) nourishes yin, activates blood, and harmonizes collaterals, ultimately restoring gastric function. Professor Tang Xudong explored the pattern identification and treatment of CAG with intestinal metaplasia based on the “Yin Fire Theory [11].” He proposed that Spleen-Stomach Qi Deficiency with Internal Yin Fire Generation represents the initial stage, treated with Modified Bu Zhong Yi Qi Tang. Internal Yin Fire Excess with Phlegm-Stasis Obstruction signifies the progressive stage, primarily managed with Modified Bu Spleen-Stomach, Drain Yin Fire, and Raise Yang Decoction. The late stage involves

spleen-kidney deficiency with mutual entanglement of phlegm and stasis, treated with modified Jianspleen-Tonify Kidney Decoction combined with Hualing Pill. This approach offers novel insights for TCM management of intestinal metaplasia. Professor Zhou summarized consensus criteria for "Northwest Dryness Syndrome" prevalent in Xinjiang, specializing in modified Seven-Ingredient Atractylodes Powder tailored to individual patterns [16]: Atractylodes macrocephala 15g, Agastache rugosa 10g, Aquilaria agallocha 9g, Pueraria lobata 20g, Fried Glycyrrhiza uralensis 6g. This combination achieves the effects of eliminating dryness, generating fluids, transforming dampness, and harmonizing the stomach. Clinical differentiation suggests that early-stage CAG often manifests as a pattern of underlying deficiency with superficial excess, primarily due to spleen-stomach deficiency and dry-dampness with blood stasis obstruction. In the progressive stage, a pattern of mixed deficiency and excess, caused by internal and external dry pathogenic factors, is more commonly observed.

In summary, contemporary medical authorities primarily treat this condition by addressing spleen-stomach deficiency, liver-stomach disharmony, internal blood stasis obstruction, and gastric yin deficiency. They further adapt their approach based on regional climatic characteristics, geographical features, and dietary habits, forming syndrome differentiation and treatment strategies tailored to local patient populations through the principle of "three factors" (climate, geography, and diet).

3.2 Physician's Self-Formulated Prescription

Professor Li Dangui, treating patients with long-standing CAG (Chronic Angina Gouty) presenting with blood stasis invading the collaterals [17], applied the theory of turbid toxins causing stasis. He employed the therapeutic principle of promoting blood circulation and resolving stasis, and clearing turbid toxins. His primary formula is the Blood-Activating and Stasis-Resolving Decoction (Angelica sinensis 9g, Ligusticum chuanxiong 9g, Lindera root 12g, Paeonia lactiflora 30g, Atractylodes macrocephala 6g, Lilium brownii 12g, Panax notoginseng powder 6g). Clinical adjustments are made based on tongue and pulse examination, gastroscopy findings, and syndrome differentiation. Professor Tian Yaozhou developed the Astragalus-Reishi Formula (Astragalus, Ganoderma lucidum [18], Cynanchum atratum seed, Coptis, Cissus quadrangularis, Curcuma, Panax notoginseng, processed Pinellia, raw oyster shell, Fructus Aurantii Immaturus, Glycyrrhiza), which clears heat, transforms dampness, strengthens the spleen, and harmonizes the stomach. Clinical application demonstrates this formula's efficacy in combating H. pylori, protecting gastric mucosa, and promoting gastric mucosal blood flow. Based on the theory of "unified qi circulation and the earth pivot governing the four phases," Xue Diankui et al. posited that CAG arises from spleen deficiency impairing transport and transformation, leading to accumulation of damp-turbid pathogenic factors [19]. They employed the modified Lizhong Tongluo Huazhuo Decoction, and combined with acupoint embedding therapy based on the Ling Shu: Commencement and Termination theory that "chronic diseases deeply penetrate and linger long." This approach achieved remarkable efficacy in treating spleen deficiency with damp accumulation-type chronic atrophic gastritis with intestinal metaplasia. Ma Xuehui et al. [20] building upon Professor Zhu Shikai's "Unblocking Therapy" and under his guidance, developed the self-formulated Fu Zheng Hu Wei Decoction. This formula, designed to tonify qi, strengthen the spleen, unblock stasis, and disperse nodules, demonstrated favorable clinical outcomes when applied to CAG treatment.

3.3 External Therapies in Traditional Chinese Medicine

A meta-analysis demonstrated that acupuncture repairs mucosal damage and delays carcinogenesis through multiple mechanisms [21]: enhancing the immune system, balancing gastrointestinal hormone levels, increasing gastric blood flow, strengthening gastric motility, regulating gastric acid secretion, reducing inflammatory responses, and controlling cell growth and apoptosis. Han Xiaojiang et al. [22] administered gastric acupuncture at three points (Zusanli, Neiguan, Zhongwan) to H. pylori-positive CAG patients alongside conventional triple therapy. Results showed significantly higher H. pylori eradication rates and overall efficacy in the observation group compared to the control group ($P < 0.05$), indicating that combining gastric acupuncture with triple therapy effectively enhances eradication rates. Zhou Yadan et al. [23-24] observed clinical symptoms, gastroscopic indicators, inflammation, and gastrointestinal hormone levels in two groups. They found that acupuncture combined with acupoint plaster application effectively alleviated clinical symptoms in CAG patients compared to conventional Western medicine treatment. The mechanism may be related to improving serum inflammatory factors and gastrointestinal hormone levels. In summary, TCM external therapies — primarily acupuncture supplemented by other modalities — can be applied singly or in combination to CAG patients with varying syndromes and constitutions. Their mechanism involves transdermal drug delivery or mechanical stimulation of acupoints to induce inflammatory responses and immune reactions, thereby regulating cytokine and gastrointestinal hormone levels. This approach improves inflammatory responses, increases gastric mucosal blood flow, and promotes mucosal repair. With diverse treatment options, significant efficacy, and and are safe and suitable. They hold great promise and value for the clinical prevention and treatment of CAG [25].

4. Mechanism Exploration

4.1 Suppression of Hp

H. pylori infection is the primary pathogenic factor driving the carcinogenic transformation of CAG gastritis. Under stress conditions, the gastric mucosa activates numerous inflammatory factors and releases large amounts of cytotoxins, triggering diverse inflammatory responses. Prolonged chronic inflammatory stimulation promotes malignant transformation of gastric mucosal tissue, ultimately leading to gastric gland atrophy [26]. Studies confirm a significant correlation between gastric cancer and precancerous lesion incidence with Hp infection, and CYP3A4 can predict the risk of progression in Hp-associated chronic atrophic gastritis [27]. Bai Haiyan et al. [28]. divided 120 Hp-positive chronic atrophic gastritis patients into two groups, administering conventional quadruple therapy and Huazhuo Detoxification

Formula for three months, respectively. The results showed that the Hp eradication rate was 73.3% with conventional quadruple therapy alone, while the eradication rate with Huazhuo Detoxification Formula intervention was 88.3%. The latter also demonstrated a lower recurrence rate and alleviated the extent of gastric mucosal damage, with its mechanism related to intervention in PGI and PGII levels. Experimental research by Li Huizhen et al. confirmed that Banxia Xiexin Decoction effectively modulates inflammatory responses in Hp-positive CAG mice, alleviating infection symptoms. Its primary mechanism involves suppressing the expression of CXCL9, CXCL10, IL-6, IL-17A, and IL-22 [29].

4.2 Modulating Inflammatory Cytokine Levels

Inflammatory responses play a central role in the development and progression of chronic atrophic gastritis (CAG). Multiple inflammatory factors, including the IL-1 family, TNF family, and NF-κB family, collectively participate in and drive the pathological process of "inflammation-to-cancer transition." In related experimental studies, Xu Yanli et al. [30] administered a modified Le Wei Yin formula to rats with CAG models and found that this formula significantly reduced gastric mucosal inflammatory damage. Its mechanism was closely associated with lowering levels of pro-inflammatory factors IL-1, IL-6, and TNF-α. Separately, Lin Xiangying et al. [31] treated CAG rats with Jianspleen Clear Heat and Resolve Stasis Decoction. Results demonstrated that this formula not only promoted gastric mucosal gland regeneration but also markedly suppressed inflammatory activity. Further studies confirmed its mechanism involves downregulating the expression of TNF-α and NF-κB signaling pathways. These studies demonstrate that TCM can mitigate mucosal inflammatory damage by multi-target regulation of the inflammatory factor network, providing crucial experimental evidence for elucidating the mechanism by which TCM reverses CAG.

4.3 Repair Damage to the Gastric Mucosa

Impaired defensive function of the gastric mucosa is a critical step in the development of chronic atrophic gastritis (CAG). Under persistent chronic inflammatory stimulation, glandular atrophy, intestinal metaplasia, and dysplasia may progressively occur, potentially leading to malignant transformation. Research indicates that molecules such as prostaglandins (PG), the TFF family (TFFs), and ADAM17 play crucial roles in regulating gastric mucosal pathology [32]. Chen Lu et al. [33] found that Diao Wei Yin effectively improved gastric mucosal pathology in CAG rats, with its mechanism related to regulating serum PGE2 and GAS levels. TFF1, acting as a gastric tumor suppressor, may activate pro-inflammatory pathways like IL-6/STAT3/NF-κB when deficient [32]. Clinical research by Tao Minghao et al. [34] demonstrated that Shenqi Taohong Tang achieved an 87.5% overall effective rate in treating CAG, potentially through regulating TFF2 levels. Furthermore, Bai Xuefeng et al. [35] experimentally confirmed that Lizhong Decoction can delay the progression of gastric mucosal atrophy, intestinal metaplasia, and dysplasia by inhibiting the ADAM17/EGFR signaling pathway, thereby exerting anti-inflammatory and mucosal repair effects. These findings collectively demonstrate that traditional Chinese medicine can delay or even reverse the inflammation-to-cancer progression in CAG by multi-targeted intervention of the aforementioned key factors and signaling pathways.

4.4 Regulation of Gastrointestinal Hormones

Gastrin (GAS) and somatostatin (SS) are crucial gastrointestinal polypeptide hormones released by endocrine cells and neurons in the digestive tract. They jointly regulate physiological processes such as gastric acid secretion, mucosal growth, and inflammatory responses. In the development of chronic atrophic gastritis (CAG), the imbalance between these two hormones is closely associated with gastric mucosal lesions. Research by Lü Xiaoyan et al. [36] indicates that Shengyang Yigai Decoction can improve gastric mucosal microcirculation disorders in CAG patients, promote glandular regeneration and repair, and further regulate gastrointestinal hormone levels. Notably, it enhances the secretion of pepsinogen II (PGII), which may represent one of its key mechanisms for reversing gastric mucosal atrophy and delaying disease progression.

4.5 Regulation of Cell Proliferation and Apoptosis

The balance between proliferation and apoptosis in gastric mucosal epithelial cells is regulated by key genes such as PCNA and Bcl-2 [37]. As a marker of cell proliferation, high PCNA expression is closely associated with gastric cancer progression and metastasis [38]. Bcl-2, an anti-apoptotic protein, participates in inhibiting programmed cell death and promoting malignant transformation. Research indicates that traditional Chinese medicine can delay CAG carcinogenesis by intervening in these factors. For instance, Qian et al. [39] found that Sini San promotes mucosal pathological repair by regulating Bcl-2 and PCNA levels. Wang et al. [40] confirmed that Bawei Shaoyao Tang suppresses PCNA overexpression, thereby mitigating gastric mucosal damage in CAG rats and halting disease progression.

5. Conclusion

In summary, traditional Chinese medicine (TCM) demonstrates significant advantages in treating chronic atrophic gastritis. Practitioners possess a deep understanding of its pathogenesis, generally recognizing that the early stage primarily involves spleen-stomach deficiency, while the middle and late stages revolve around the intertwined progression of deficiency, stagnation, stasis, and toxicity. Regarding treatment, current approaches largely rely on individualized empirical formulas, limiting their scalability and lacking unified standards for efficacy evaluation. The mechanisms of action for some herbal medicines remain at the hypothesis stage, with molecular pathways and target points yet to be clearly defined. Future efforts should systematically compile the experience of renowned practitioners to establish foundational formulas that can be modified based on syndrome differentiation. This should be combined with individualized treatment considering factors such as geography, diet, and age. Additionally, research into molecular and genetic mechanisms should be strengthened, and the integration of Chinese and Western medicine should be deepened to enhance clinical applicability and efficacy.

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