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Study on the Relationship between Distribution of Traditional Chinese Medicine Syndromes and Pathological Changes in Chronic Atrophic Gastritis

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Abstract

Objective: This study aims to investigate the distribution of traditional Chinese medicine (TCM) syndrome types in chronic atrophic gastritis (CAG) and explore the correlation between TCM syndrome types and pathological changes, providing an objective basis for clinical syndrome differentiation and treatment. Methods: A retrospective study was conducted, involving the summary and analysis of medical records from 201 CAG patients who met the inclusion criteria. TCM syndrome differentiation was performed, followed by logistic regression analysis of the relationship between TCM syndrome types and pathological changes in the patients' gastric mucosa. Results: The distribution of TCM syndrome types among the 201 patients, from highest to lowest, was liver-stomach heat stagnation syndrome, liver-stomach "Qi" stagnation syndrome, spleen-stomach damp-heat syndrome, spleen-stomach weakness syndrome, stomach collaterals stasis syndrome, and stomach "Yin" deficiency syndrome. There was a statistically significant difference in the distribution of intestinal metaplasia among different TCM syndrome types. Compared to the liver-stomach "Qi" stagnation syndrome, the liver-stomach heat stagnation syndrome showed a significant increase in mild intestinal metaplasia ($p < 0.05$). No statistically significant differences were observed in the distribution of inflammation, activity, glandular atrophy, and dysplasia among different TCM syndrome types. In the correlation analysis between syndrome types and pathological changes, the liver-stomach "Qi" stagnation syndrome was negatively correlated with intestinal metaplasia, while the spleen-stomach damp-heat syndrome was positively correlated with glandular atrophy. The remaining syndrome types showed no correlation with pathological changes. Conclusion: A certain correlation is observed between TCM syndrome types of CAG and pathological changes of gastric mucosa .

Keywords

Chronic atrophic gastritis

Traditional Chinese medicine syndrome types

Pathological changes

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1. Introduction

Chronic atrophic gastritis (CAG) is a chronic gastric disease characterized by atrophy of the intrinsic glandular tissue, with or without intestinal metaplasia and dysplasia. Its clinical manifestations are often atypical, with common symptoms including discomfort, fullness, dull pain, and burning pain in the upper and middle abdomen, as well as dyspeptic symptoms such as loss of appetite, belching, acid regurgitation, and nausea. Traditional Chinese medicine classifies it under conditions such as "stomach ache", "indigestion with rumbling stomach", "acid regurgitation", and "distention and fullness".

Chronic atrophic gastritis is closely associated with the development of gastric cancer. Traditional Chinese medicine offers unique advantages in the diagnosis and treatment of CAG, effectively improving clinical symptoms, slowing disease progression, and reducing the risk of carcinogenesis. Previous domestic studies have shown a certain correlation between pathological changes in the gastric mucosa of CAG and TCM syndrome types, but a unified consensus has not been reached among the research findings [1,2]. This study employs a retrospective research method to explore the correlation between the distribution of TCM syndrome types and pathological changes in CAG, providing an important reference for the clinical diagnosis and treatment of this disease.

2. Materials and methods

2.1. Case sources

The clinical and pathological data from 201 cases (confirmed as chronic atrophic gastritis through endoscopic biopsy with complete case data) was collected in this study, at Suining Hospital of Traditional Chinese Medicine from July 2023 to December 2024, including 104 males and 97 females, aged 19–90 years (average age 61.1 years).

2.2. Diagnostic criteria

2.2.1. Western medicine diagnostic criteria

The clinical and pathological diagnostic criteria for chronic atrophic gastritis refer to the "Consensus on Chronic Gastritis in China (2017, Shanghai)" [3].

2.2.2. Traditional Chinese medicine syndrome differentiation criteria

The diagnostic criteria and syndrome type judgment for traditional Chinese medicine refer to the "Consensus on Integrated Traditional Chinese and Western Medicine Diagnosis and Treatment of Chronic Atrophic Gastritis (2017)" [4]. There are six main syndrome types of Chronic Atrophic Gastritis (CAG), including liver-stomach "Qi" stagnation syndrome, liver-stomach stagnant heat syndrome, spleen-stomach weakness syndrome (spleen-stomach deficiency-cold syndrome), spleen-stomach damp-heat syndrome, stomach "Yin" deficiency syndrome, and stomach collateral stasis syndrome.

2.3. Inclusion and exclusion criteria

2.3.1. Inclusion criteria

(1) Meet the Western medical diagnostic criteria for CAG and the diagnostic criteria for TCM syndrome differentiation
(2) Agree to undergo endoscopic pathological biopsy.

2.3.2. Exclusion criteria

(1) Acute gastritis with symptoms such as vomiting, hematemesis, abdominal pain, or diarrhea
(2) Presence of tumors such as gastric or duodenal ulcers, gastric cancer, or esophageal cancer
(3) Long-term use of hormones, non-steroidal anti-inflammatory and analgesic drugs, anti-rheumatic drugs, anti-tumor drugs, or undergoing chemotherapy
(4) Pregnant or lactating women
(5) Concomitant conditions such as heart failure, cirrhosis with portal hypertension, diabetes, uremia, or severe hepatitis.

2.4. Research methodology

A retrospective research approach was employed, involving the retrieval of medical records that met the diagnostic criteria for CAG. Patient information, including basic demographics, clinical symptoms, tongue and pulse conditions, as well as endoscopic and pathological data, was collected. Two associate senior physicians or higher determined the TCM syndrome types based on TCM syndrome differentiation criteria.

2.5. Statistical analysis

Statistical analysis was performed using SPSS 22.0 software. Continuous variable data were expressed as mean ± standard deviation ($\bar{x} \pm s$).

Basic statistical descriptions of qualitative data utilized frequency tables, percentages, or composition ratios.

Non-normally distributed data were described using maximum values, minimum values, and medians. Categorical variables in univariate analysis were tested using the χ^2 test. The correlation between gastric mucosal pathological changes and TCM syndrome types was analyzed using logistic regression. Hypothesis testing employed one-sided tests, with $\alpha = 0.05$ set as the significance level.

3. Results

3.1. The distribution of TCM syndrome types in CAG

The frequency of distribution of various TCM syndrome types in CAG, from highest to lowest, is as follows: liver-stomach stagnation-heat syndrome at 36.82% (74/201) > liver-stomach "Qi" stagnation syndrome at 23.38% (47/201) > spleen-stomach damp-heat syndrome at 16.42% (33/201) = spleen-stomach deficiency syndrome at 16.42% (33/201) > stomach meridian stasis syndrome at 4.97% (10/201) > stomach "Yin" deficiency syndrome at 1.99% (4/201) (refer **Table 1**).

Table 1. Distribution of TCM syndrome types in patients with chronic atrophic gastritis

TCM Syndrome Type	Cases (n)	Percentage (%)
Liver-stomach "Qi" stagnation	47	23.38%
Spleen-stomach damp-heat	33	16.42%
Liver-stomach stagnant heat	74	36.82%
Spleen-stomach deficiency	33	16.42%
Stomach collateral blood stasis	10	4.97%
Stomach "Yin" deficiency	4	1.99%

3.2. Distribution of different syndrome types in gastric mucosal inflammation, activity, glandular atrophy, intestinal metaplasia, and dysplasia

3.2.1. Distribution of different syndrome types in gastric mucosal inflammation and activity

There is no statistically significant difference in the distribution of inflammation and activity among different syndrome types, as shown in **Table 2**.

3.2.2. Distribution of different syndrome types in gastric mucosal glandular atrophy, intestinal metaplasia, and dysplasia

There is no statistically significant difference in the distribution of glandular atrophy and dysplasia among different syndrome types. However, there is a statistically significant difference in the distribution of intestinal metaplasia among different syndrome types ($p = 0.032$), as shown in **Table 3**. Using pairwise Fisher's exact test,

Table 2. Distribution of different syndrome types in gastric mucosal inflammation and activity

TCM Syndrome Type	n	Inflammation		Activity		
		Mild	Moderate-Severe	None	Mild	Moderate-Severe
Liver-stomach "Qi" stagnation	47	28	19	30	15	2
Liver-stomach stagnant heat	74	43	31	38	31	5
Spleen-stomach damp-heat	33	22	11	20	12	1
Spleen-stomach deficiency	33	17	16	19	10	4
Stomach collateral blood stasis	10	6	4	6	2	2
Stomach "Yin" deficiency	4	2	2	1	3	0
χ^2-value		1.72		8.39		
p-value		0.886		0.591		

compared to the liver-stomach "Qi" stagnation syndrome, the liver-stomach stagnation-heat syndrome showed a significant increase in mild intestinal metaplasia (p = 0.033).

3.3. Correlation between TCM syndrome types and pathological changes

3.3.1. Correlation analysis between liver-stomach "Qi" stagnation syndrome and pathological changes

Logistic regression analysis was conducted with "liver-stomach "Qi" stagnation syndrome" as the dependent variable and activity, Hp, inflammation, glandular atrophy, intestinal metaplasia, and dysplasia as independent variables. The results indicate a negative correlation between liver-stomach "Qi" stagnation syndrome and intestinal metaplasia (OR = 0.094, p = 0.001), as shown in **Table 4**.

3.3.2. Correlation analysis between liver-stomach damp-heat syndrome and pathological changes

Using "liver-stomach damp-heat syndrome" as the dependent variable and activity, Hp, inflammation, glandular atrophy, intestinal metaplasia, and dysplasia as independent variables, a logistic regression analysis was conducted. The results indicated no correlation between liver-stomach damp-heat syndrome and pathological changes, as shown in **Table 5**.

3.3.3. Correlation analysis between spleen-stomach damp-heat syndrome and pathological changes

Using "spleen-stomach damp-heat syndrome" as the dependent variable and activity, Hp, inflammation, glandular atrophy, intestinal metaplasia, and dysplasia as independent variables, a logistic regression analysis was conducted. The results indicated a positive correlation between spleen-stomach damp-heat syndrome and

Table 3. Distribution of different syndrome types in gastric mucosal glandular atrophy, intestinal metaplasia, and dysplasia

TCM syndrome type	n	Glandular atrophy		Intestinal metaplasia			Dysplasia		
		Mild	Moderate-severe	None	Mild	Moderate-severe	None	Low-grade	High-grade
Liver-Stomach "Qi" Stagnation	47	36	11	7	29	11	46	1	0
Liver-Stomach Stagnant Heat	74	62	12	1	61	12	72	0	2
Spleen-Stomach Damp-Heat	33	20	13	1	22	10	32	1	0
Spleen-Stomach Deficiency	33	24	9	2	22	9	30	2	1
Stomach Collateral Blood Stasis	10	9	1	0	9	1	9	1	0
Stomach Yin Deficiency	4	2	2	0	2	2	4	0	0
χ^2-value		13.31		19.7			8.78		
p-value		0.207		0.032*			0.553		

Table 4. Correlation analysis between liver-stomach "Qi" stagnation syndrome and pathological changes

Pathological indicator	B	SE	Wald χ^2	df	p	OR	95% CI
(Intercept)	1.303	0.742	3.081	1	0.079	3.680	(0.859, 15.770)
Activity	-0.030	0.376	0.007	1	0.936	0.970	(0.464, 2.028)
Inflammation	-0.064	0.340	0.035	1	0.851	0.938	(0.482, 1.828)
Hp	-0.822	0.419	3.854	1	0.050	0.440	(0.193, 0.999)
Intestinal metaplasia	-2.368	0.740	10.247	1	0.001	0.094	(0.022, 0.399)
Glandular atrophy	-0.012	0.395	0.001	1	0.976	0.988	(0.456, 2.142)
Dysplasia	-0.796	1.085	0.539	1	0.463	0.451	(0.054, 3.781)

Table 5. Correlation analysis between liver-stomach damp-heat syndrome and pathological changes

Pathological indicator	B	SE	Wald χ^2	df	p	OR	95% CI
(Intercept)	-2.185	1.068	4.187	1	0.041	0.113	(0.014, 0.912)
Activity	0.498	0.318	2.454	1	0.117	1.645	(0.883, 3.065)
Inflammation	0.043	0.301	0.020	1	0.887	1.044	(0.579, 1.882)
Hp	-0.347	0.330	1.103	1	0.294	0.707	(0.370, 1.351)
Intestinal metaplasia	1.609	1.068	2.270	1	0.132	4.998	(0.616, 40.540)
Glandular atrophy	-0.693	0.374	3.439	1	0.064	0.500	(0.240, 1.040)
Dysplasia	-0.650	0.836	0.604	1	0.437	0.522	(0.101, 2.688)

Table 6. Correlation analysis between spleen-stomach damp-heat syndrome and pathological changes

Pathological indicator	B	SE	Wald χ^2	df	p	OR	95% CI
(Intercept)	-0.545	0.223	5.954	1	0.015	0.580	(0.374, 0.898)
Activity	0.470	0.320	2.155	1	0.142	1.599	(0.854, 2.994)
Inflammation	-0.446	0.407	1.200	1	0.273	0.640	(0.288, 1.422)
Hp	-0.223	0.338	0.435	1	0.510	0.800	(0.412, 1.552)
Intestinal metaplasia	-0.599	0.379	2.502	1	0.114	0.549	(0.261, 1.154)
Glandular atrophy	0.918	0.407	5.085	1	0.024	2.503	(1.128, 5.558)
Dysplasia	-0.249	1.093	0.052	1	0.820	0.780	(0.091, 6.646)

Table 7. Correlation analysis between spleen-stomach deficiency syndrome and pathological changes

Pathological indicator	B	SE	Wald χ^2	df	p	OR	95% CI
(Intercept)	-2.122	1.072	3.916	1	0.048	0.120	(0.015, 0.980)
Activity	0.053	0.408	0.017	1	0.896	1.055	(0.474, 2.347)
Inflammation	0.391	0.388	1.015	1	0.314	1.479	(0.691, 3.165)
Hp	-0.187	0.426	0.193	1	0.661	0.829	(0.360, 1.911)
Intestinal metaplasia	0.561	1.076	0.272	1	0.602	1.753	(0.213, 14.446)
Glandular atrophy	0.273	0.438	0.388	1	0.534	1.314	(0.557, 3.101)
Dysplasia	1.291	0.769	2.820	1	0.093	3.637	(0.806, 16.412)

Table 8. Correlation analysis between stomach collateral stasis syndrome and pathological changes

Pathological indicator	B	SE	Wald χ^2	df	p	OR	95% CI
(Intercept)	-2.859	0.457	39.203	1	<0.001	0.057	(0.023, 0.140)
Activity	-0.203	0.700	0.084	1	0.772	0.816	(0.207, 3.220)
Inflammation	15.726	2061.32	0.000	1	0.994	6753252.872	(0.000, Inf)
Hp	0.240	0.701	0.117	1	0.733	1.271	(0.321, 5.022)
Intestinal metaplasia	0.026	0.672	0.002	1	0.969	1.026	(0.275, 3.828)
Glandular atrophy	-1.017	1.070	0.902	1	0.342	0.362	(0.044, 2.949)
Dysplasia	0.998	1.137	0.770	1	0.380	2.713	(0.292, 25.190)

glandular atrophy (OR = 2.503, p = 0.024), as shown in **Table 6**.

3.3.4. Correlation analysis between spleen-stomach deficiency syndrome and pathological changes

Using "spleen-stomach deficiency syndrome" as the dependent variable and activity, Hp, inflammation, glandular atrophy, intestinal metaplasia, and dysplasia as independent variables, a logistic regression analysis was conducted. The results indicated no correlation between spleen-stomach deficiency syndrome and pathological changes, as shown in **Table 7**.

3.3.5. Correlation analysis between stomach collateral stasis syndrome and pathological changes

Using "stomach collateral stasis syndrome" as the dependent variable and activity, Hp, inflammation, glandular atrophy, intestinal metaplasia, and dysplasia as independent variables, a logistic regression analysis was conducted. The results indicated no correlation between stomach collateral stasis syndrome and pathological changes, as shown in **Table 8**. However, due to the occurrence of intestinal metaplasia in all cases of stomach collateral stasis syndrome included, leading to inter-group imbalance, the OR value and standard error were excessively large.

4. Discussion

The etiology of CAG is complex, often resulting from factors such as exogenous pathogenic factors, improper diet, emotional distress, and spleen-stomach deficiency, which damage the spleen and stomach, leading to dysfunction in their ascending and descending functions, as well as disrupted "Qi" flow in the middle jiao, ultimately causing the disease. Clinically, this condition predominantly manifests as a combination of root deficiency and branch excess, with intermingled deficiency and excess syndromes. The root deficiency primarily involves spleen-stomach weakness and stomach "Yin" deficiency, while the excess pathologies mainly consist of "Qi" stagnation and blood stasis, as well as damp-heat. Chao Jun and colleagues explored the evolution pattern of TCM syndromes inCAG and found that during the transition from non-atrophic to atrophic gastritis, intestinal metaplasia, and dysplasia, there exists a syndrome evolution from excess to deficiency, with the gradual emergence of "Yin" deficiency and blood stasis [5]. Lu Xiaojie and other scholars believed that in the pathological evolution of gastric mucosa in CAG precancerous lesions, patients with mild pathological changes predominantly exhibit excess syndromes, while those with moderate to severe pathological changes mainly present with a mix of deficiency and excess syndromes [6]. Wan Yuchen analyzed and suggested that spleen-stomach weakness is the fundamental cause of the disease, with liver "Qi" stagnation and spleen-stomach damp-heat serving as intermediate developmental stages [7]. As spleen deficiency and liver stagnation progress, blood circulation becomes impaired, or damp-heat steams and scorches, leading to blood stasis and obstruction of the gastric meridians. Furthermore, prolonged blood stasis and obstruction can injure "Yin", resulting in stomach "Yin" deficiency.

This study, through statistical analysis of the distribution of TCM syndrome types in 201 patients with CAG, found that liver-stomach stagnation-heat syndrome and liver-stomach "Qi" stagnation syndrome are the primary syndrome types of chronic atrophic gastritis, followed by spleen-stomach damp-heat syndrome, spleen-stomach weakness syndrome, gastric meridian blood stasis syndrome, and stomach "Yin" deficiency syndrome. The analysis suggests that CAG may often be caused by liver "Qi" stagnation, impaired spleen function, liver "Qi" transforming into fire, scorching stomach "Yin", and damp turbidity obstructing the Middle Jiao, ultimately leading to syndromes such as stagnation-heat, deficiency-heat, and damp-heat [8]. Zhu Yanan found in her survey that liver-stomach disharmony syndrome is the most predominant TCM syndrome type, which is generally consistent with the findings of this study [9]. Zhang Ruifen, by summarizing the recent research on CAG, discovered that liver "Qi" invading the stomach syndrome and spleen-stomach weakness syndrome have the highest prevalence rates, followed by other syndrome types such as gastric meridian blood stasis, stomach "Yin" deficiency, liver-stomach "Qi" stagnation, spleen-stomach damp-heat, and liver-stomach stagnation-heat [10].

The results of this study showed no statistically significant differences in the distribution of activity,

inflammation, glandular atrophy, and dysplasia among different syndrome types. However, there were significant differences in the distribution of intestinal metaplasia among different syndrome types. Compared with the liver-stomach "Qi" stagnation syndrome, the mild intestinal metaplasia in the liver-stomach stagnation-heat syndrome increased significantly. In the correlation analysis between syndrome types and pathological changes, the liver-stomach "Qi" stagnation syndrome was negatively correlated with intestinal metaplasia, while the spleen-stomach damp-heat syndrome was positively correlated with glandular atrophy. No correlation was found between the remaining syndrome types and pathological changes. The author speculated that in the early stage of atrophic gastritis, the liver-stomach "Qi" stagnation syndrome predominated. As mild intestinal metaplasia appeared in the gastric mucosa, the liver-stomach stagnation-heat syndrome increased significantly, which was consistent with the evolution of TCM pathogenesis of "Qi" stagnation transforming into heat over time". Previous scholars have proposed that "damp-heat" was involved in the entire process of gastritis-to-cancer transformation. The persistence of damp and heat pathogens, which were difficult to resolve, was also an important reason for the continued presence of a chronic inflammatory microenvironment. Professor He Xiaohui [11] believed that damp-heat was the key to initiating CAG, and collateral stasis was the pathological key. The disease was located in the spleen and stomach and was also closely related to the liver and gallbladder in regulating dispersion and dispersal function and the renal insufficiency. Cheng Ruodong and others found that glandular atrophy and intestinal metaplasia of the gastric mucosa were strongly correlated with the spleen-stomach deficiency syndrome, while dysplasia of the gastric mucosa was strongly correlated with the stomach collateral stasis syndrome [2]. Another study statistically analyzed the distribution of TCM syndrome types in elderly patients with moderate to severe intestinal metaplasia and found that the syndromes of stomach "Yin" deficiency and stomach collateral obstruction were more prevalent [12].

This study found that the four syndrome types with the highest proportions were liver-stomach stagnation-heat syndrome, liver-stomach "Qi" stagnation syndrome, spleen-stomach damp-heat syndrome, and spleen-stomach deficiency syndrome. Thus, it can be seen that factors such as stagnation-heat, "Qi" stagnation, damp-heat, and deficiency played crucial roles in the onset and progression of this disease. In the correlation analysis between syndrome types and pathological changes, a certain correlation was observed between the pathological changes of the gastric mucosa and TCM syndrome types in CAG. The liver-stomach "Qi" stagnation syndrome was negatively correlated with intestinal metaplasia, while the spleen-stomach damp-heat syndrome was positively correlated with glandular atrophy. No correlation was found between the remaining syndrome types and pathological changes. In clinical practice, the application of microscopic syndrome differentiation combined with the traditional four diagnostic methods of TCM provides a reliable basis for the early detection and treatment of diseases to intercept their progression. Due to the limitations of time and sample size in this study, fewer patients with the stomach "Yin" deficiency syndrome were included. Therefore, it is necessary to increase the sample size for further in-depth research to provide a more credible reference for the syndrome differentiation and treatment of CAG.

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Disclosure statement

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