

FACTORS ASSOCIATED WITH CHRONIC GASTRITIS IN PATIENTS WITH PRESENCE AND ABSENCE OF HELICOBACTER PYLORI

Fatores associados com a gastrite crônica em pacientes com presença ou ausência do Helicobacter pylori

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ABSTRACT - Background - Chronic gastritis is an inflammation of the stomach mucosa, which is considered its main etiological factor the *Helicobacter pylori*. **Aim** - To observe the differences in patients with chronic gastritis as well as the presence and absence of *H. pylori*, to obtain a better understanding of the etiological factors, clinical, dietary and lifestyle habits and associated diseases. **Methods** - This was a descriptive study, retrospective medical records of patients with chronic gastritis treated as outpatients. Endoscopy and research of *H. pylori* was used in the diagnosis of chronic gastritis. The survey was conducted through a questionnaire to ascertain the risk factors for chronic gastritis and the clinical manifestations of disease, dietary and lifestyle habits, family history of the disease, weight changes and medications. For statistical analysis was used Spearman coefficient. Data were considered statistically significant $p < 0.05$. **Results** - Of the 94 patients evaluated were symptomatic with heartburn, belching, epigastric pain, fullness and nausea. In 56.6% (n = 54) of individuals was detected the presence of bacteria and in 43.6% (n = 40) was not found any specific etiologic factor. Was performed chi-square statistical test of clinical manifestations and factors such as stress and anxiety, leading to positive correlation. It became evident that the risk factors for disease are diverse, including the use of drugs, alcoholism, eating quickly, talking during meals, with significant relation to abdominal distension and reflux. The patients with the bacteria, had a higher risk of experiencing these symptoms. **Conclusion** - Various etiological factors in eating habits and lifestyle, as smoking, alcoholism, anxiety, stress, associated diseases and inadequate nutrition, interact to the onset of clinical manifestations, and the presence and absence of *H. pylori* did not show significant changes in patient clinical status.

HEADINGS - Gastrointestinal diseases. Nutrition. Diet therapy.

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Financial source: none
Conflicts of interest: none

Received for publication: 11/10/2011
Accepted for publication: 16/02/2012

RESUMO - Racional - A gastrite crônica é inflamação da mucosa do estômago, que tem como principal fator etiológico o *Helicobacter pylori*. **Objetivo** - Verificar fatores associados com a gastrite crônica em pacientes com presença e ausência do *H. pylori*, visando obter maior conhecimento sobre os fatores etiológicos, manifestações clínicas, hábitos alimentares e de vida nesses pacientes. **Métodos** - Trata-se de um estudo descritivo, retrospectivo com dados de prontuário de pacientes com gastrite crônica atendidos em ambulatório. A pesquisa foi realizada por meio de um questionário que investigava fatores etiológicos da gastrite crônica, bem como as manifestações clínicas das doenças, os hábitos alimentares e de vida, entre outros. Endoscopia digestiva e pesquisa do *H. pylori* foi a forma de diagnóstico da gastrite crônica. Para análise estatística foi utilizado o teste qui-quadrado. **Resultados** - Dos 94 pacientes avaliados a maioria era sintomático, apresentando pirose, eructações, dor epigástrica, plenitude gástrica e náuseas. Em 56,6% (n=54) dos indivíduos foi detectada a presença da bactéria e em 43,6% (n=40) não foi achado fator etiológico específico. Os resultados que se mostraram significativos foram em relação à distensão abdominal e refluxo gastroesofágico, observando-se aumento desses fatores em pacientes que tinham a bactéria. Além disso, tornou-se evidente que além do *H. pylori* outros fatores estão relacionados com a gastrite crônica como: utilização de medicamentos, tabaco, álcool e pacientes que apresentavam hábitos alimentares com alimentação inadequada, realização de refeições rápidas, e dialogar durante as refeições. Também foram verificados estarem relacionados ansiedade, estresse e doenças associadas que pudessem aumentar a secreção ácida. **Conclusão** - Vários fatores etiológicos oriundos de hábitos alimentares e estilo de vida, como tabagismo, alcoolismo, ansiedade, estresse, doenças associadas e nutrição inadequada, interagem para o início das manifestações clínicas, e a presença ou ausência de *H. pylori* não mostrou diferenças significativas no estado clínico dos pacientes.

DESCRITORES - Gastroenteropatias. Nutrição. Dietoterapia.

INTRODUCTION

Chronic gastritis is inflammation of stomach mucosa that has as main causal factor the *Helicobacter pylori* with great prevalence (about 50% in world population). Has universal distribution and increases with age¹⁰.

One in each six individuals infected by it has risk of developing ulcer; 1-3% in USA and 12% in Japan have chances to develop cancer of stomach during lifetime⁴.

Having in view the epidemiology, the infection has a typical interpersonal standard of transmission, with intense prevalence in young people in developed countries and also in populations of low income. The index of acquisition has decreased in developed countries and elderly have more prevalence than the young people, due to better hygiene^{4,17}.

The infection by *Helicobacter pylori* in relationship to its prevalence, varies between developed and in development countries - less us first -, but independently of regions the differences are based in socioeconomic opportunities of population¹¹.

Its not defined exactly the reason about the relationship of *Helicobacter pylori* and gastritis; however, it is believed that is predetermined by socioeconomic factors, environmental and cultural practices, beyond genetics possible predisposition. Several studies suggest that the genetic factor has less influence that the ones related to housing⁶.

There are several Brazilian studies on the infection by *H. pylori*^{2,7,12}. The main prevalence is : 59.5% on Rio de Janeiro (RJ); 76.3% in São Paulo (SP); 83% in Santa Maria (RS); 84.7% in Nossa Senhora do Livramento (MT); 85.18% in Botucatu (SP); 87% in Araçuaí (MG); 89.6% in Campinas (SP) and 96% in São Luis (MA)⁷.

Beyond of *H. pylori* there are several factors that can be involved on development of chronic gastritis. The chronic use of alcoholic beverages by example, can cause erythema and erosions, and the injuries produced by intake of alcohol result on disruption of barrier of gastric mucosa and consequently, the backscatter of ions H^{+} ¹⁷.

Numerous studies show that the alcohol and drugs are harmful agents to the gastric mucosa and episodes of reflux can also produces lesion¹⁴.

In summary, the etiological factors are inappropriate diet, smoking, alcoholism, drugs and intake of corrosive substances, stress, trauma, surgical procedures, septicemia, liver failure, irradiation upon the stomach, systemic infections and also the *H. pylori*¹⁹.

The present study has as goal to check the clinical factors associated with the diagnosis of chronic gastritis and the presence or absence of *H. pylori*.

METHODS

This study was approved by the Ethics Committee of the institutions under protocol n. 057.2011.2, following the guidelines and standards regulatory of research involving human beings contained in Resolution No 196/96.

It is descriptive retrospective with data from patients suffering of chronic gastritis in Santa Maria, RS, Brazil. The collection was held on period from March to May, 2011.

The inclusion criteria were patients above 18 years that attend the clinic of gastroenterology and had the diagnosis of gastritis after digestive endoscopy.

The of exclusion criteria were patients with associated diseases of gastrointestinal tract.

The research was based in a questionnaire that looked for the causal factors of chronic gastritis on first treatment, as well as the clinical manifestations of the disease, the food habits and lifestyle, history of the disease in the family, changes on weight and use of drugs. The data were tabulated on program SPSS and were considered statistically significant when $p < 0.05$ (chi-square).

RESULTS

Were included 94 patients with chronic gastritis with age average of 48 ± 16 years and 67.8% (N = 61) women and 32.2% (N = 33) men.

In the digestive endoscopy was based the diagnosis of chronic gastritis and to check if the causal agent could be the *H. Pylori*. This bacterium was present in 56.6% (N = 54) of patients and 43.6% (N = 40) did not have a causal specific agent. However, factors as smoking, alcoholism and quick ingestions of meals were involved in pathogenesis. From the total, 16% (N = 15) were asymptomatic.

In relationship to patients that had symptoms, the clinical main manifestations were heartburn in 40.5% (N = 38), belching in 43.6% (N = 40), nausea in 33% (n = 31), vomiting in 9.6% (N = 9) and 55.3% (N = 52) abdomen pain. The episodes of diarrhea occurred in 10.6% (N = 10) causing loss of weight depending on the duration and intensity; anorexia was present in few patients (4% N = 4).

Intestinal habit was normal in 58.5% (N = 54). Constipation was the complain presented by the others.

All these prior factors were not significant among them ($P > 0.05$) and with the existence or not of *H. pylori*. However, abdominal distension and gastroesophageal reflux differed statistically in the patients that had *H. pylori* in relationship to the ones that did not have it. Presence of bacterium had 3.5 times more risk of present abdominal distension and four times more risk of present gastroesophageal reflux (Table 1). The presence of typical symptoms of gastritis was marked in first query.

TABLE 1 – Factors related with the chronic gastritis in patients with presence and absence of *H. pylori*

Exposure	Helicobacter pylori		ODDS not adjusted	Chi-Square	P
	Presence N (%)	Absence N (%)			
Heartburn	P	19 (35,2)	0,6 (0,26 – 1,38)	1,44	0,16
	A	35 (64,8)			
Eructation	P	17 (31,5)	0,3 (0,13 – 0,72)	7,59	0,05
	A	37 (68,5)			
Nausea	P	15 (27,8)	0,57 (0,24 – 1,37)	1,55	0,15
	A	39 (72,2)			
Vomiting	P	4 (7,4)	0,56 (0,14 – 2,23)	0,68	0,31
	A	50 (92,6)			
Abdominal pain	P	29 (53,7)	0,85 (0,37 – 1,95)	0,13	0,43
	A	25 (46,3)			
Abdominal distension	P	36 (66,7)	2,21 (0,95–5,11)	3,47	0,04
	A	18 (33,3)			
Gastroesophageal reflux	P	08 (14,8)	6,78 (0,81– 56,63)	4,02	0,04
	A	46 (85,2)			
Intestinal habit preserved	P	28 (51,9)	0,51(0,22– 1,21)	2,31	0,09
	A	26 (48,1)			
Diarrhea	P	6 (11,1)	1,12(0,29– 4,28)	0,03	0,57
	A	48 (88,9)			
Asymptomatic	P	8 (14,8)	0,82 (0,27– 2,48)	0,12	0,47
	A	7 (17,5)			

ODDS: IC of 95%; P = Presence; A = Absence.

Food intolerance was observed in 48.9% (N = 46) patients and related to fatty bean dairy products, peppers, marked spicy food, eggs and alcoholic beverages.. Fifty and eight percent of patients (N = 55) reported to eat very quickly. Beyond this 50% (N = 47) reported to dialogue during the meals. Drinking liquids during meals was present in 62.8% (N = 59) and 58.6% (N = 55) respectively in relationship to patients with and without the bacteria. All these circumstances did not have statistic difference ($p > 0.05$) when compared the existence or not of *H. pylori* (Table 2).

Forty and one patients (43.6%) reported to ingest tea daily in several times. The difficulty in mastication and swallow was reported by 8.5% (N = 8) mainly in the old people. Tobacco was referred by 26.6% (N = 25) and alcohol abuse in 20.2%

TABLE 2 – Food habits in patients with chronic gastritis and presence or not of *H. pylori*

Exposure	Helicobacter pylori		ODDS not adjusted	Chi-Square	P
	Presence N (%)	Absence N (%)			
Drinking on meals	P	36 (66,7)	1,47 (0,63 – 3,43)	0,82	0,24
	A	18 (33,3)			
Difficulties on mastication	P	4 (7,4)	0,72 (0,16 – 3,07)	0,19	0,46
	A	50 (92,6)			
Changes in body weight	P	6 (11,1)	0,70 (0,21 – 2,38)	0,31	0,39
	A	48 (88,9)			
Eating quickly	P	34 (63,0)	1,53 (0,67 – 3,53)	1,03	0,21
	A	20 (37,0)			
Dialogue during meals	P	28 (51,9)	1,19 (0,52 – 2,69)	0,17	0,41
	A	26 (48,1)			
Mate/tea	P	23 (42,6)	0,90 (0,39–2,06)	0,54	0,49
	A	31 (57,4)			

ODDS: IC of 95%; P = Presence; A = Absence.

(N = 19). Thirty five patients (37.4%) had some degree of stress and 60 (64%) were considered with anxiety and with change of food habits. The anxious patients usually ate quickly. Only 6% (N = 6.4) reported to do self-medication, such as anti-inflammatory drugs, corticosteroids and aspirin. All these factors did nor have significant

TABLE 3 – Lifestyle of patients with chronic gastritis and presence or absence of *H. pylori*

Exposure	Helicobacter pylori		ODDS not adjusted	Chi-Square	P
	Presence N (%)	Absence N (%)			
Smokers	P	15 (27,8)	1,15 (0,45 – 2,92)	0,09	0,47
	A	39 (72,2)			
Alcoholic abuse	P	14 (25,9)	2,45 (0,80 – 7,48)	2,56	0,08
	A	40 (74,1)			
Stress	P	21 (38,9)	1,18 (0,50 – 2,76)	0,14	0,43
	A	33 (61,1)			
Drugs	P	4 (7,4)	1,52 (0,26 – 8,73)	0,22	0,49
	A	50 (92,6)			
Anxiety	P	36 (66,7)	1,33 (0,57 – 3,11)	0,44	0,32
	A	18 (33,3)			

ODDS: IC de 95%; P = P; A = Ausência.

differences statistically ($p > 0.05$) when compared with the existence of *H. pylori* (Table 3).

In relationship to change of weight only 12.7% (N = 12) had it related to episodes of vomiting, diarrhea or simply by looking for the ideal weight. All patients did not refer familiar history of chronic gastritis nor previous operations on gastrointestinal tract.

From all patients, 18% (N = 17) said to have obesity. Also, 10% (N = 10) reported to have diabetes and 16 (17%) hypertension, with no statistic differences ($p > 0.05$) when compared with the existence or not of *H. Pylori* (Table 4).

TABLE 4 – Diseases associated with presence or absence of *H. pylori*

Exposure	<i>Helicobacter pylori</i>		ODDS not adjusted	Chi-Square	P
	Presence N (%)	Absence N (%)			
Obesity	P	10 (18,5)	1,07 (0,36 – 3,1)	0,01	0,56
	A	44 (81,5)			
Diabetes	P	6 (11,1)	1,12 (0,29 – 4,28)	0,03	0,57
	A	48 (88,9)			
Hypertension ¹	P	6 (11,1)	0,37 (0,12 – 1,13)	3,13	0,06
	A	48 (88,9)			

ODDS: IC de 95%; P = P; A = Ausência.

DISCUSSION

Although there are many causal factors to trigger chronic gastritis, infection by *H. pylori* is important one. It colonizes the gastric mucosa and establishes chronic infection highly associated with gastritis¹⁶. So, the endoscopy has fundamental place in defining diagnosis. It induces infection and shares with the syphilis and the tuberculosis certain characteristics in common¹⁰. The infection normally is acquired in childhood and adults tend to contaminate themselves through their children⁴.

Many studies tried to explain the mechanisms that are considered essential to the success of *H. pylori* in colonize the stomach, a very hostile environment. Although predominant, the presence of *H. pylori* is not the only factor. Environmental and genetics pre-disposition of patients also contribute^{1,5}.

The transmission seems to happen by contact between person / person, fecal / oral or oral / oral, and also by water contamination^{1,5,17}.

Food interfere and can change gastric motility. A very hot intake leads to congestion of mucosa and raises the secretion of acid and decreases the time of evacuation^{13,15}.

Alcoholic beverages stimulate acid secretion. The soft drinks decrease the pressure beneath the esophageal sphincter and can produce gastroesophageal reflux¹³. The nicotine also decreases this pressure and induces hyperchloridia. The spicy food increase the gastric secretion and cause constant irritations in the mucosa. The red pepper and paprika raise the acid secretion. The black pepper causes irritation raising secretions and producing dyspepsia. The chilli pepper and mustard cause the erythema and gastric lesion¹³.

The broths with large quantities of purine are exciting of gastrointestinal mucosa and act raising the acid secretion. The concentrated carbohydrates resulting in stimulation of osmoreceptors, and act on retardation of stomach deflation. Food with high levels of fat act on retardation of gastric

emptying¹³.

The intolerances generate dyspeptic symptoms, as fullness, belching, heartburn, nausea and precocious satiety.

The relationship of alcohol with the prevalence of gastritis and the infection by *H. pylori* already was studied in alcoholic patients; the bacterium was found in 14 of 18 individuals. After four weeks of abstinence, there was no changes in histology, demonstrating that the alcohol can not be considered the main agent⁸. Its routine intake leads to increase of resistance of mucosa caused by high synthesis of endogenous prostaglandin¹⁰.

Psychosomatic factors or environmental stimuli can act stimulating or inhibiting the gastric motility. The probability of patients with gastritis and presence of *H. pylori* having abdominal distension, is more likely to happen with the absence of bacteria.

The anti-inflammatory drugs are very used today and decrease the defensive factors mainly decreasing submucosal blood flow, causing ischemia¹⁰.

The lack of time currently is a great factor to bad digestion, leading the people to eat quickly and dialogue during meals. In a study on asymptomatic patients with diagnosis of chronic gastritis had the presence of *H. pylori*; it was concluded that the infection occurs in stomach of people apparently normal and increase in prevalence with age³.

Studies have established that feed balance, rich in fruits and vegetables, rich in antioxidants, as vitamins C and E and carotenoids, are related with the decrease of gastric lesion^{5,18}.

CONCLUSION

Various etiological factors in eating habits and lifestyle, as smoking, alcoholism, anxiety, stress, associated diseases and inadequate nutrition, interact to the onset of clinical manifestations, and the presence and absence of *H. pylori* did not show significant changes in patient clinical status.

REFERENCES

1. Aguiar DCF. Expressão dos antígenos ABH e Lewis na gastrite crônica e alterações pré-neoplásica da mucosa gástrica. *Arq Gastroenterol*. São Paulo. 2002; 39 (4): 222-232.
2. BresCiani C, Latif I, Coser RB, Yagi O, Deutsch CR, Mucerino D, Zilberstein B, Cecconello I. Determinação histopatológica da presença do *helicobacter pylori* em câncer gástrico. *ABCD Arq Bras Cir Dig*. 2011;24(1):59-63
3. Cornelius P, Dooley MD, Cohen MDH, Patrick L, Fitzgibbons MD, Bauer M, et. al. Prevalence of *Helicobacter pylori* Infection and Histologic Gastritis in Asymptomatic Persons. *NEJM*. California,1989; 321:1562-1566.

4. Graham D. Tratado de Medicina Interna. São Paulo, Saunders Elsevier, 2005.
5. Izzotti A, Durando P, Ansaldia F, Gianiorio F, Pulliero A. Interaction between *Helicobacter pylori*, diet, and genetic polymorphisms as related to non-cancer diseases. *Mutation Research*, 2009; 667: 142–157.
6. Kodaira M. Aspectos epidemiológicos do *Helicobacter pylori* na infância e adolescência. *Rev Saúde Pub, São Paulo*. 2002; 36 (3): 356-369.
7. Ladeira, MSP. Biopatologia do *Helicobacter pylori*. *J Bras Patol Med Lab*, 2003; 39(4): 335-342.
8. Lieber C. Gastritis in the alcoholic: Relationship to gastric alcohol metabolism and *Helicobacter pylori*. *Food Rev Intern*. 1998; 4: 423-433.
9. MINCIS, M. Gastroenterologia e Hepatologia. São Paulo, Lemos: 1: 276-287; 1997
10. Miszputen S. Gastroenterologia. São Paulo, Manole, 2007.
11. Moraes M. Fatores de risco para infecção pelo *Helicobacter pylori* em crianças. *J Ped*, Rio de Janeiro. 2003; 79 (1): 21-28.
12. Petroianu A, Rocha CG, Alberti LR, Costa AMC. Estudo morfológico macro e microscópico da gastrite cáustica em ratos. *Rev Col Bras Cir*. 2001;28(6):404-407
13. Reis N. Nutrição Clínica. Sistema digestório.1ª ed. Rio de Janeiro: Rubio, 2003.
14. Rubin E. Patologia. Bases clinicopatológicas da medicina. Guanabara Koogan S.A, Rio de Janeiro, 2006
15. Salehi, Z. The relationship between *Helicobacter pylori* infection and gastric adenocarcinoma in Northern Iran. *Onc res*, Iran, 2010.
16. Telford JL, Ghiara P, Dell'orco MM, Comanducci D, Burrioni M, Bugnoli MF, et al. Gene structure of the *Helicobacter pylori* cytotoxin and evidence of its key role in gastric disease. *The J Exp Med*, 1994; 179 (5): 1653-1658.
17. Vergueiro CV, Cordioli R, Martucci D, Peres V, Kiyamu A, Ribeiro KCB, Chiattone CS. Soroprevalência e fatores associados à infecção pelo *Helicobacter pylori* em doadores de medula óssea de São Paulo. *Rev Bras Epidemiol São Paulo*; 2008 11(2): 196-203.
18. Zojaji H, Talaie R, Mirsattaria D, Haghazalia M, Molaei M, Mohseniana N, Derakhshana F, Zali MR The efficacy of *Helicobacter pylori* eradication regimen with and without vitamin C supplementation *Digestive and Liver Dis*, 2009; 41: 644–647.