Histopathological Study of Chronic Gastritis in GIT Center at Babylon

Haider Abdul Ridha Kehaiwish
Department of Pathology, College of Medicine, University of Babylon.Hilla, Iraq.

Abstract

Back ground: Chronic gastritis defined as histopathological entity that characterized by chronic inflammation of the gastric mucosa. There are three different types can be distinguished: gastritis A the result of an autoimmunological process with antibodies against parietal cells and intrinsic factor and is confined to the proximal stomach. Distal, antral gastritis B which is caused by an infection of the gastric mucosa with H. pylori. Gastritis **C** may be caused by drugs and alcohol but is mainly found in gastric remnants after partial resection as a consequence of biliary reflux.

Aim: To give a detailed histolopathological study of chronic gastritis with and without intestinal metaplasia and relationship between H pylori infection and chronic gastritis at GIT center at Babylon according to the Modified Sydney system.

Methods: Gastroscopic biopsy specimens from 50 consecutive patients from GIT center at Babylon were examined histologically after staining with hematoxylin /eosin and modified giemsa stain and graded according to the Updated Sydney System for H. pylori infection, chronic inflammation, neutrophil activity, glandular atrophy and intestinal metaplasia.

Results: The rate of the graded variables, in the antrum and corpus respectively, were as follows: H pylori infection (82%, 78 %), chronic inflammation (98%, 78%), neutrophil activity (70%, 62%), glandular atrophy (12%,8%) and intestinal metaplasia (11%, 5%). Significant association was found between the degree of H pylori colonization with chronic inflammation, less degree of neutrophil activity and antral glandular atrophy. Biopsies from the antrum and corpus showed significant histopathological discordance for all the graded variables.

Conclusion: The study shows that the majority of patients show a mild to moderate degree of inflammation with or without intestinal metaplasia, still less degree of glandular atrophy some time present. The study confirms that H pylori is the chief cause of gastritis in the study patients. The study also shows that interrelationships between the histological variables in those patients are similar to those found in other populations worldwide.

دراسة باثولوجية نسيجية لالتهاب المعدة المزمن في مركز الجهاز الهضمي في بابل

الخلاصة

خلفية البحث:التهاب المعدة المزمن يعرف كونه التهاب الطبقة المخاطية المبطنة للمعدة بشكل مز من، هنالك ثلاث أنواع من هذا الالتهاب :نوع(A) والمحدد بمنطقة جسم المعدة والناتج عن مرض مناعة ذاتي ضد خلايا المعدة الجدارية والعامل الداخلي، نوع (B) والمحدد بالمنطقة البعيدة لتجويف المعدة والناتج عن عدوى بكترية ال H.pylori، ونوع (C) الناتج عن الأدوية، الكحول واسترجاع مادة الصفراء.

الهدف من الدراسة: دراسة التهاب المعدة المزمن بشكل مفصل من حيث المسببات والتغيرات المصاحبة للالتهاب المزمن كالتحول المعوي لبطانة المعدة ونشاط الإلتهاب المتمثل بالخلايا البلعمية وعلاقة التهاب المعدة المزمن بالإصابة ببكتريا ال ***H. pylori***.

المرضى والمواد وطرق العمل

أحذت خزع نسيجية بواسطة تنظير المعدة من ٥٠ مريض في مركز الجهاز الهظمي في بابل .فحصت العينات نسيجيا بعد صبغها بصبغة هيماتوكسلين والأيوسين وصبغة كمزا المعدلة ودُرجت حسب نظام سدني المحدث للمتغيرات الآتية (الأصابة ببكتريا ***H. pylori*** (٨٢%،٧٨%، الألتهاب المزمن(٩٨%،٧٨%)، نشاط الخلايا البلعمية، ضمور الغدد المعدية والتحول المعوي) .

النتائج: النسب لدرجة المتغيرات التي تم دراستها لمنطقة الفتحة البوابية وجسم المعدة كانت حسب الترتيب الأتي: الإصابة ببكتريا ال H. Pylori ٨٢%،٧٨%، التهاب المعدة المزمن ٩٨%،٧٨%، تغلغل الخلايا البلعمية بنسبة ٧٠%،٦٢%، ضمور الغدد المعوية بنسبة ١٢%،٨%و التحول المعوي ١١%،٥%.

الاستنتاجات: أظهرت الدراسة أن هنالك علاقة مهمة بين الإصابة ببكتريا H. pylori والتهاب المعدة المزمن، تغلغل الخلايا البلعمية وضمور بطانة المعدة كما أظهرت الدراسة بأن هنالك اختلاف في جميع المتغيرات التي تم دراستها بين جسم المعدة ومنطقة الفتحة البوابية بما يتماشى مع نتائج لعدة دراسات أجريت في مناطق أخرى في العالم.

Introduction

Chronic gastritis is defined as the presence of chronic mucosal inflammatory changes leading eventually to mucosal atrophy and epithelial metaplasia[1]. H. pylori infection is the most common cause of type B chronic gastritis. The disease most often presents as a predominantly antral gastritis with high acid production, despite hypogastrinemia. The risk of duodenal ulcer is increased in these patients and, in most; gastritis is limited to the antrum with occasional involvement of the cardia[2]. In a subset of patients the gastritis progresses to involve the gastric body and fundus. This pangastritis is associated with multifocal mucosal atrophy, reduced acid secretion, intestinal metaplasia, and increased risk of gastric adenocarcinoma [3-5]. The mechanisms by which H. pylori cause gastritis are incompletely defined, it is clear that infection results in increased acid production and disruption of normal gastric and duodenal protective mechanisms, H. pylori gastritis is, therefore, the result of an imbalance between gastroduodenal mucosal defenses and damaging forces that overcome those defenses[6-8].

The prevalence of H pylori, a worldwide infection, varies greatly among countries and among population groups within the same country [9]. Once acquired in childhood, this bacterium is able to establish a life-long relationship with its host10. The epithelium responds to infection by mucin depletion, cellular exfoliation, and compensatory regenerative changes. Polymorph infiltration into foveolar and surface epithelium, and lamina propria edema are conspicuous. Collections of polymorphs in the foveolae and adherent neutrophil exudate on the surface may also be present. In the few cases of acute Helicobacter gastritis there is relatively equal involvement of antrum and body[11,12]. This acute phase is accompanied by profound hypochlorhydria and a failure of ascorbic acid secretion into gastric juice [13,14]. It may take several weeks for acid output to return to preinfection levels, and in a proportion of patients output remains low. However, ascorbic acid secretion remains lower than normal for the duration of chronic gastritis, indicating

that low secretion is related to persisting inflammation rather than hypochlorhydria [13]. Over time chronic antral H. pylori gastritis may progress to pangastritis, resulting in multifocal atrophic gastritis[15,16]. The underlying mechanisms contributing to this progression are not clear, but interactions between the host and bacterium seem to be critical. Polymorphisms in TNF and a variety of other genes associated with the inflammatory response influence the clinical outcome in H. pylori infection[18]. Severity of disease may also be influenced by genetic variation among H. pylori strains.

Other less common types of gastritis include type A gastritis that confined to the proximal stomach and is the result of an autoimmunological process with antibodies against parietal cells and intrinsic factor. It is a rare disorder and may lead to pernicious anaemia. Gastritis type C may caused by drugs and alcohol but is mainly found in gastric remnants after partial resection as a consequence of biliary reflux [10].

Morphological changes in gastric biopsy specimens generally demonstrate H. pylori in infected individuals. The organism is concentrated within the superficial mucus overlying epithelial cells in the surface and neck regions. The distribution can be irregular, with areas of heavy colonization adjacent to those with few organisms [8]. In extreme cases, the organisms carpet the luminal surfaces of foveolar and mucous neck cells, and can even extend into the gastric pits. Organisms are most easily demonstrated with a variety of special stains like modified Giemsa stain and immunohisto-chemistry[18]. H. pylori not found in association with gastric intestinal metaplasia or duodenal epithelium. However, H. pylori may be present in foci of pyloric metaplasia within chronically injured duodenum or gastric-type mucosa within Barrett esophagus. Endoscopic finding in H. pylori–infection usually there is erythematous antral mucosa with coarse or nodular appearance associated with intense inflammation. The microscopical examination reveal inflammatory infiltrate with variable numbers of neutrophils within the lamina propria, including some that cross the basement membrane to an intraepithelial location and accumulate in the lumen of gastric pits to form pit abscesses. In addition, the superficial lamina propria includes large numbers of plasma cells and increased numbers of lymphocytes and macrophages. Intraepithelial neutrophils and subepithelial plasma cells are characteristic of H. pylori gastritis [19].

Grading of the gastritis is useful in evaluation of the severity of chronic gastritis. The modified Sydney system describes practical guidelines for grading histopathological features of the gastritis by using visual analogue scale20. The Sydney System for the classification of gastritis emphasized the importance of combining topographical, morphological, and etiological information into a schema that would help to generate reproducible and clinically useful diagnoses. Modified Sydney System were established to take an agreed terminology of gastritis; to identify, define, and attempt to resolve some of

the problems associated with the Sydney System[21, 22]. Overall, the principles and grading of the Sydney System were only slightly modified, the Sydney system made to grade histological parameters, identify topographical distribution and, finally, make a statement about the etiopathogenesis of the gastritis. Of pathogenetic importance is, in the first instance, the differentiation between gastritis with and gastritis without H pylori infection. The group of H pylori-associated gastritis can be further subdivided into forms of gastritis whose morphological distribution patterns usually identify them as sequelae of H pylori infection, while the group of gastritis unassociated with H pylori, can be differentiated into autoimmune, chemically induced reactive gastritis, ex. Crohn's gastritis and a number of special forms of gastritis. The aim of this study was to assess those patients complaining from chronic gastritis histopathologically depending on modified Sydney grading system.

Patients, Material and Methods

The study was conducted in the endoscopy unit of GIT center at Babylon city. A prospective descriptive study that performed on 50 consecutive adult patients complaining from symptoms of chronic gastritis, admitted to this center from the period between January to April 2011, gastric endoscopic biopsies were done by endoscopy (*Olympus250F, Japan*) and biopsies were taken from the corpus and antrum of the stomach. These biopsies were taken from corpus and antrum of the stomach then fixed with 10 % formalin. After usual processing for each biopsy; 2 sections of 5 ?m thickness sections from their corresponding paraffin blocks. The assessment was done on H&E stained sections by using the Sydney System for graded variables of gastritis. Using an Olympus BX 20. Modified-Giemsa stained sections were used for H pylori assessment.

Statistical analysis

Chi-square test and Mann-Whitney *U* test were used to analyze differences and compare variables between various groups. $P < 0.05$ was considered statistically significant. SPSS version 15 was used for statistical analysis.

Result

In this study gastric biopsies from 50 patients with chronic gastritis that met the criteria were analyzed. The number of patients with moderate and severe inflammation was small.

Total of 42 cases (84%) had histological evidence of *H pylori* infection, 42 (84%) antrum infection and 34 (68%) corpus infections. Most of the cases showed a mild degree of colonization by *H pylori* (24%) and (60%) for corpus and antrum biopsies respectively. Severe, H.P. colonization was seen in (6)12%. Only 18 cases (36%) were *H pylori* negative with neutrophil activity in the absence of *H pylori*. Significant association was found between the degree of *H pylori* colonisation and chronic inflammation in the corpus and antrum as well as between *H pylori* colonisation, neutrophil activity and inflammation $P < 0.001$. Glandular atrophy in the antrum was seen in 9 cases (18%), of them 7 (14%) were of mild degree and 2 (4%) of moderate degree. Severe

atrophy was not seen. Glandular atrophy in the corpus was seen in 8 cases (16%). Regarding intestinal metaplasia, five cases 5 (10%) were seen in the corpus and 10 (20%) in the antral area. Only 3 cases showing intestinal metaplasia were of moderate degree and there is no case of severe intestinal metaplasia was noted, there is no significant correlation between H.P.infection, glandular atrophy and intestinal metaplasia (P=0.5).

Table 1 Show the scoring percentage:

Variable	Score	Negative (0)	Mild (1)	Moderate (2)	Severe (3)
Chronicity	Corpus Antrum	11(22%) 1(2%)	30(60%) 9(18%)	7(14%) 18(36%)	2(4%) 22(44%)
Activity	Corpus Antrum	19(36%) 15(30%)	13(26%) 8(16%)	15(30%) 17(34%)	3(6%) 10(20%)
Atrophy	Corpus Antrum	42(92%) 41(88%)	6(12%) 7(14%)	2(6%) 2(4%)	0 0
Intestinal Metaplasia	Corpus Antrum	45(90%) 40(80%)	4(8%) 8(16%)	1(2%) 2(4%)	0 0
H.pylori	Corpus Antrum	18(36%) 6(12%)	12(24%) 30(60%)	8(16%) 10(20%)	2(4%) 4(8%)

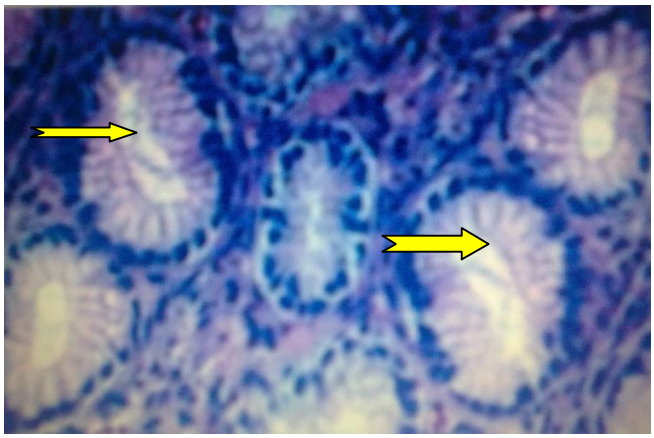

Figure1: Photomicrograph show H.pylori infection;H&E stain X 400

Figure2: Photomicrograph show intestinal metaplasia associated with H.pylori infection;H&E stain X 100

Discussion

It is well known that the most important etiologic association with chronic gastritis is chronic infection by the bacillus H. pylori. The histopathological results from this study provide further evidence that *H pylori*-associated gastritis is the most common cause of chronic gastritis among adult patients presenting to GIT center at Babylon city this results

agree with another study in Iraq[22]. The morphological assessment of the gastric mucosa with increased rate of *H pylori* detection by modified Giemsa stain that increase the sensitivity of histological procedure for diagnosing *H pylori* infection. The findings in this study confirm that *H pylori* are causally associated with chronic inflammation, neutrophil activity and mild degree of glandular atrophy in the stomach[19]. The findings are similar to those reported from results elsewhere around the world[21]. In similar to other study this study also reveal that gastritis in those patients is mainly antral-predominant with significant discordance in the severity of graded variables between antral and corpus biopsies [22]. The majority of patients undergoing endoscopy in this setting were seen to have moderate to severe gastritis, but much lower rates and severity of glandular atrophy and intestinal metaplasia. These findings are in agreement with the results from other study in Iraq and some study around the world [23,24].

In this study, the prevalence of intestinal metaplasia was lower in the H pylori-positive group. Although intestinal metaplasia is a virtually constant component of atrophic gastritis, and is found more frequently in the stomach of patients with *H pylori* gastritis[19,20] in patients with extensive antral intestinal metaplasia, a type of epithelium to which *H pylori* rarely adheres the infection is virtually confined to the non metaplastic areas of the corpus [24]. In this study direct observation of the microorganism by microscopy confirms *H. pylori* infection and the frequency of active gastritis among these patients was 70%, this result similar to the results from other areas in the Middle East region [24].

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