Helicobactor pylori Infection and Gastric Carcinoma in Babylon Governorate

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Abstract

The study aimed to determine the relationship between H. pylori & gastric ca.The period of the study covered 24 months and started in Nov. 1997. 37 patients with gastric ca. entered into the study and the diagnosis of gastric carcinoma was made by clinical examination and confirmed by upper GIT endoscopy and histopathological examination. Also eighty four subjects were taken as healthy control. Three antral biopsy specimens were taken , inaddition to biopsy from the lesion, and these specimens were subjected to the three main important methods, including :
Urease test
Microbiological culture
Histopathological changes in the gastric mucosa .
The results show that 30 out of 37 patients (8 1.1 %) has got evidence of H. pylori infection associated with sever atrophy of gastric glands, intestinal metaplasia and marked inflammatory cells & polymophs infiltrations in the intraepithelial regions and lamina properia . The control group shows only 19 % positive evidence of H. pylori infection .

الخلاصه

هدفت هذه الدراسة الى معرفة العلاقة بين بكتريا H.pylori ومرض القرحة الهضمية . فترة الدراسة كانت 24 شهر حيث ابتدأت في الشهر الحادي عشر لعام 1997 تم دراسة حالة 37 مريض من المرضى المصابين بسرطان المعدة والذين . تم تشخيص حالتهم بواسطة الفحوصات السريرية .فحص الناظور والفحوصات النسيجية المرضية وادخل في هذه الدراسة 84 من الاصحاء كمجموعة مقارنة . تم اخذ ثلاث خزع من غاز المعدة بالاضافة الى خزعة واحدة من منطقة الاصابة واجريت على هذه الخزع فحص اليوريز الزرع البكتيري والتقطيع النسيجي . نتأج هذه الدراسة بينت ان 30 مريض من اصل 37 مريض (81,1 %) كانو مصابين ببكتريا H.pylori وكان لديهم ضمور شديد بالغدد المعدية تحول قسم من الخلايا المعدية لديهم الى خلايا معوية وترشيح شديد للخلايا الالتهابية والخلايا متعددة اشكال النوى في منطقة النسيج الطلائي والصفيحة القاعدية . اما بالنسبة الى مجموعة السيطرة فكان 19% فهم يحمل بكتريا H. pylori

Introduction

H. pylori (H p) infection is most common where socioeconomic condition is poor ; and it is a prime candidate for an early life influence on risk of stomach cancer; given that persistent infection is often acquired in childhood . (1- 4)

The proposed sequence for development of gastric adenocarcinoma was well correlated to H p infection of

gastric mucosa and Hp infection is the main environmental factor causing active chronic atrophic gastritis with loss of chief cells & parietal cells and making a media susceptible to the effects of carcinogens , which are generated by an over growing nitrifying bacteria and some other dietary factors , including high salts intake & low vitamin C consumption .(5)(6)

The rates of stomach cancers are declining rapidly in many countries, especially in the developed world partly might be due to a corresponding decline in the prevalence of H p infection. (7) (8) Also primary gastric lymphoma & the mucosa associated lymphoid tissue lymphoma (MALT) have been associated with H p infection . (9)
The (MALTOMA) type has been shown to regress after eradication of H p in about 80 % of cases .(10)

The most accurate methods(11) of diagnosis of H p infection are based on performing of multiple diagnostic tests , which may not be universally available ;and two categories of tests are present including :
(a) Endoscopic (invasive)
(b) Non- endoscopic (non – invasive)
The tests for which endoscopies are not necessary include various methods of antibody detection and the carbon-labeled urea breath tests & also the newly-developed immunoassay that detects bacterial antigens in a faecal specimens (12)(13)(14)(15)(16) .The serological tests (17) (18) can provide a reliable non-invasive methods for antibody detection of H p infection & it shows sensitivity from 95.5% for ELISA technique to 80% for Latex Agglutination test; and the specificity range from 76.9% in ELISA to 69.2% by Latex Agglutination test .

We did a study in order to detect the relation of H p infection & gastric Ca. in our patients and the main methods used for the diagnosis , are rapid urease tests, microscopy of tissue specimen & bacteriological culture .

Patients & Methods

We made a study in the endoscopy unit of Mirjan hospital over a period of about 2 years from Nov. 1997 to May 2000.We enrolled in this study 37 patients with gastric Ca. & 21 are males and 16 are females; their mean ages equal to(58.5 years).We also include in this study 84 control subjects .Upper GIT endoscopy were performed on allthese patients using Olympus-type PQ endoscope and its various accessories. We perform tissue biopsy by using endoscopy forceps& usu. we take three antral biopsy and these specimens were subjected to urease test , bacteriological culture methods on blood ager plates and under micro-aerophilic condition and last sample was tested for histopathological changes which could be seen under light microscopy .
The urease test was considered positive , if gastric antrum mucosal biopsy specimen was placed in sterile plastic tube containing the test reagent and the test was judged to be positive if a colour had occurred including a change in colour of the sample from yellow to red within 24 hrs time .(19)

During histopathological assessment of gastric mucosal biopsy specimens , the specimens were fixed in pouins solution & stained with haematoxylin & eosin stain. The histopathological changes & grading of its severity were assessed by two pathologists and according to Sydney system . (20) Patients with two positive results were considered infected with Hp.

Results

The following tables demonstrate the detection rates of H p infection in patients with gastric Ca.

Table 1 demonstrate the detection rates of H. pylori in different tests

Table 1

Types of test	No.	Positive	%	Negative	%
Urease test	73	31	83.8	6	16.2
Culture	73	25	67.5	2	32.5
Histopathology	73	30	81.1	7	18.9

Table (2) Demonstrate the comparison between control groups and patients with gastric carcinoma and detection rate of H. pylori infection , which was statistically significant in patient with gastric carcinoma.

Table 2

Subject	**No**	**Number of patient infected**	**Percentage**
Control group	**84**	**16**	**19%**
Patients	**37**	**30**	**81%**

In figure number 1 we demonstrate the histopathological differences in antral gastric mucosa between patient with gastric ca infected with Hp and patient with gastric ca . non—infected with Hp and from this figure it seems that the market inflammatory cells and polymorphs cells infiltration in the gastric gland, intestinal metaplasia with infected with Hp shows atrophy of the lamina properia and intra epithelial mucosa of the gastric antrum whereas tha mucosa of the gastric antrum of patient non-infected showed mild atrophy of gastric gland and mild intestinal metaplasia and little cells infiltration.

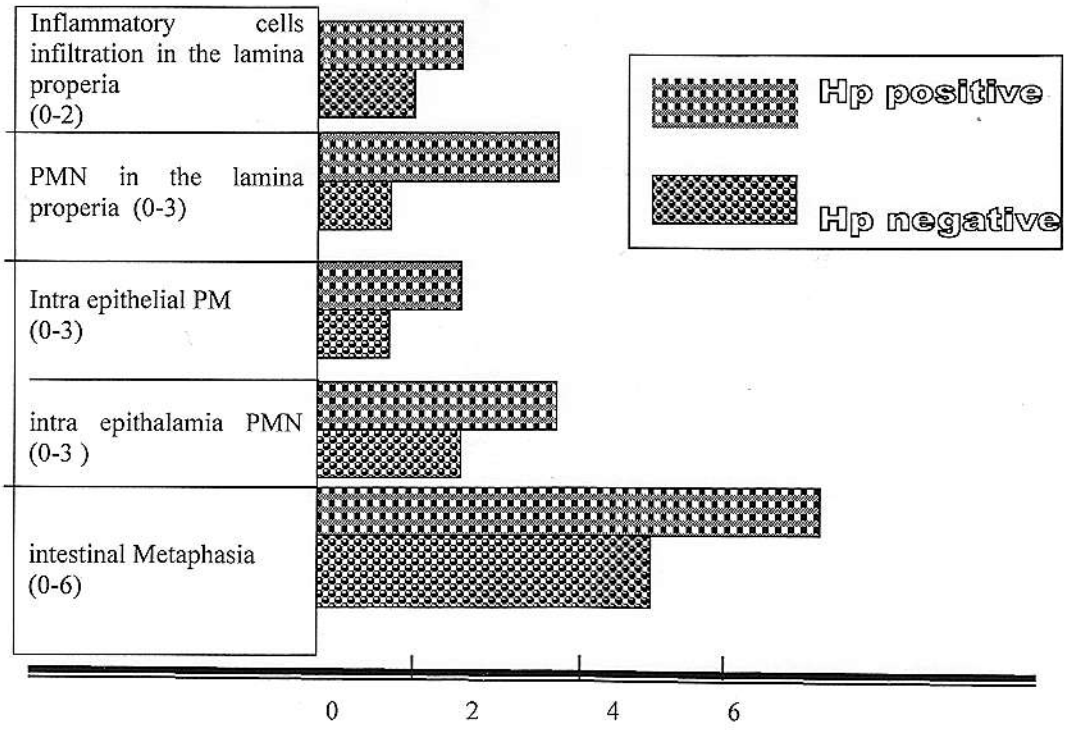

Figure 1 : The Histopathological Differences Between Hp Positive And Hp Negative Patient

Discussion

There is now strong epidemiological evidence that Hp infection is associated with the development of gastric adenocarcinoma . (21) The association between H pylori infection and stomach cancer rises public health questions about how to reduce the burden of childhood infection and whether this in- turn reduce the incidence of stomach cancer needs further evaluation . (22)

A number of mechanisms have been postulated to explain how Hp infection pre disposes to gastric lesion and cancer (23),(24) The leading hypothesis is that the increased cancer risk is due to induction of an inflammatory response .

It is possible that chronic Hp infection lead to chronic atrophic gastritis with the resulting achlorhyderia which in turn favors bacterial over growth that can convert nitrates in dietary components to nitrites . These nitrites in combination with genetic factors promote abnormal cellular proliferation and eventually cancer (25) (26)

For that reason Hp has led to cultural revolution in the field of Gastroenterology with a tendency toward considering this bacterium as the culprit of many gastric disorders .

Hp undoubtedly represents a new and relevant etiological factor in the carcino genic sequence of Correa(4 ; 5) being responsible for the onset of chronic gastritis . The existing differences between the prevalence of the infection and that of atrophic gastritis is difficult to explain if other variable such as the genotypic characteristic of the individual and environmental factors are not taken into accounts .

There are few points in the literature that totally question the relationship between Hp and gastric cancer . (27) One point is that the prevalence of Hp is the same in both; in the various countries examined , while the male to female ratio for gastric ca. points to a

greater frequency in males .The 2nd point is based on data from several developing countries and in the study documented by Massirno Crespi that the prevelance of H p infection is as high as 90% with very low frequency of gastric cancer in these countries , but the argument on this ; is that the onset of cancer could be related to the acquisition of the infection which is particularly high in younger age groups .The 3rd point that should be raised is the fact that Hp is responsible for duodenal ulcer (D U) and carriers of DU present a low risk of gastric ca., however , only the aggressive strains of Hp , that are producer of Cag A toxins(18) could play an important role in carcinogenesis of gastric Ca.

In conclusion : The relation of H p infection & cancer could be explained under the following headings

a-The role of H p should be related to only the initial promoter phase of inflammation , while the subsequent events are secondary to many other variables.

a- The possible role of H p infection appears to be directed more toward undermining the defense capability of gastric mucosa , makes it more susceptible to the environmental carcinogens or irritants .

b- In developed countries the incidence of gasric ca. low ; this is probably due to reduction in the rate of Hp infection and this would emphasize the importance of H p eradication in the prevention of gastric ca.

c- We need to prepare ourselves for a difficult period in which careful use of antibiotics currently available to eradicate H p infection is warranted so as to prevent emergence of H p antibiotic resistance . (28)

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