Immunoglobulin G and immunoglobulin M anti-*Helicobacter pylori* antibodies—diagnostic and prognostic value in gastritis

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INTRODUCTION

Helicobacter pylori is a gram-negative, curved bacillus thought to play role in the pathogenesis of certain gastroduodenal diseases like non-ulcer dyspepsia (NUD), chronic inflammation, ulcers, etc. The *H. pylori* bacteria reside in the mucous layer covering the gastric epithelium, and therefore the type of protective measures that could confer resistance appear to be limited. Although, *H. pylori* infection stimulates strong local and systemic specific immunoglobulin (Ig) A and IgG antibody production, the influence of antibodies on bacterial colonisation and gastric inflammation is still debatable.¹

MATERIALS AND METHOD

We investigated 30 patients of NUD. Informed consent was taken and antral biopsies were performed using standard upper gastrointestinal endoscopes (Olympus Optical Co Ltd., Tokyo, Japan). Three biopsies were taken and processed by standard methods. Biopsies were evaluated using a modification of the Sydney system.² About 4 mL of peripheral blood was collected from the patients and the serum was separated. Serum samples stored at –20°C were tested for the presence of IgG and IgM antibodies against *H. pylori* using standard enzyme-linked immunosorbent assay (ELISA) (Genesis diagnostics, UK). Eight controls were run with each set of samples. In addition to the positive and negative control, six serial dilutions of the antigen were included. A standard curve was constructed based upon the optical density (OD) of the controls. The OD of the test samples were then read off the curve. Repeat ELISA's were done as and when required.

The morphological parameters against which the IgG and IgM anti-*H. pylori* levels were measured were the density of various inflammatory cells like neutrophils, eosinophils, lymphocytes, and plasma cells. The IgG and IgM antibody titres were also compared with the grade of atrophy, the presence or absence of intestinal metaplasia, and glandular shortening. Lastly and most importantly, the titres were also compared with the presence and density of *H. pylori*.

RESULTS

Thirty patients were evaluated. The minimum IgG titre recorded was 0.111 and the maximum titre was 2.118 IU/L. The mean IgG titre was 0.846 IU/L and the median titre was 0.655 IU/L. The minimal IgM titre recorded was 0.424 IU/L and the maximum titre was 2.437 IU/L. The mean titre was 1.046 IU/L and the median titre was 0.837 IU/L.

No correlation between the various inflammatory parameters and the IgG and IgM antibody titres and no correlation between the antibody titres and the presence and grade of atrophy, gland shortening, and intestinal metaplasia was observed. In conclusion, we noted that there was no correlation between the antibody titres and the presence and density of *H. pylori* in the antral biopsies. Table 1 shows the *P* value between the antibody titres and the various parameters.

DISCUSSION

This small pilot study did not find any correlation between the IgG and IgM titres and the severity of gastritis as assessed by histopathology. A few studies have found that there is a significant correlation between the severity of gastritis and the antibody titres.³ Yamamoto et al found that serum anti-*H. pylori* IgG antibody titres were correlated significantly with the severity of inflammation in both the antrum and body. Significant associations were found between serum anti-*H. pylori* IgG and IgA antibody titres and the development of atrophic gastritis. They did not find a correlation with anti-*H. pylori* IgM titres. They concluded that their results suggest that measurement of serum anti-*H. pylori* antibody titres is

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Table 1 The correlation between the various inflammatory parameters and the IgG and IgM titres.

IgG	IgM	Neutrophils	Lymphocytes	Gland atrophy	Intestinal metaplasia	Fibrosis	Plasma cells	Eosinophils	Lymphoid follicles	Activity	<i>H. pylori</i>
0.716	1.007	0	1	0	0	0	1	0	0	0	0
1.631	0.6	1	2	0	0	1	2	0	2	0	1
1.461	2.353	0	1	1	0	1	1	0	0	0	0
0.417	1.329	0	1	1	0	1	1	0	3	0	0
0.118	0.883	1	1	1	0	1	1	0	0	0	2
0.586	1.773	0	2	1	0	1	2	0	2	1	0
0.312	0.563	0	2	1	0	2	2	0	0	0	0
1.371	1.001	0	2	1	0	1	2	0	0	0	0
2.118	0.653	0	3	1	0	2	2	0	0	0	0
0.881	2.237	1	3	1	0	1	2	0	0	0	2
0.143	0.424	1	3	1	0	1	3	0	2	1	2
0.118	0.883	1	1	2	0	2	1	0	3	0	2
0.691	1.201	0	2	2	0	2	2	0	0	0	0
1.811	0.54	1	2	2	0	2	2	0	0	1	2
0.011	2.437	0	3	2	0	2	2	0	3	0	0
1.238	0.757	1	3	2	0	2	3	0	2	0	1
0.655	0.791	1	1	3	0	3	1	0	0	0	0
0.37	1.008	2	2	3	0	2	2	1	2	1	2
0.398	0.622	3	2	3	0	2	2	0	0	1	3
0.533	0.637	0	3	3	0	3	2	0	0	0	0
1.569	0.683	2	2	3	0	3	3	0	2	1	2
0.155	1.231	0	3	3	0	3	3	0	0	0	0
1.642	0.738	1	3	1	1	2	2	0	0	0	0
1.953	0.519	1	3	2	1	2	2	1	2	0	1
1.739	0.701	1	3	1	2	1	3	0	0	0	1
0.404	1.134	2	3	2	2	2	3	0	2	0	2
0.863	1.394	2	3	2	2	2	3	0	0	1	3
0.2	1.65	1	2	3	2	3	2	0	2	1	2
0.438	0.566	2	2	0	3	1	1	0	0	1	2
IgG and inflammatory parameters (<i>P</i> values)		0.853	0.248	0.237	0.917	0.670	0.377	0.487	0.214	0.454	0.426
IgM and inflammatory parameters (<i>P</i> values)		0.106	0.759	0.998	0.740	0.456	0.469	0.478	0.835	0.601	0.388

H. pylori: *Helicobacter pylori*, IgG: immunoglobulin G, IgM: immunoglobulin M.

useful in the diagnosis of *H. pylori* infection and severity of gastritis.

Other studies deny such a correlation. Gerencser et al concluded that serology does not always parallel the macroscopic pictures revealed by gastroscopy.⁴ However, the authors also felt that serology could play an important role in follow-up tests and examinations as well as in understanding the aetiology of different gastroduodenal diseases.

On the basis of a survey and of previous studies performed by them, Czinn et al concluded that at a serum dilution of 1:800, IgG but not IgA or IgM antibody to *H. pylori* is useful in

the rapid screening of symptomatic children for the presence of *H. pylori*.⁵ This is an observation that has been contested by Akhiani who suggested that antibodies are not only dispensable for protection, but they impair both the elimination of bacteria and the development of gastritis.¹ This effect appears to be IgA-dependent and is not a function of specific IgM or IgG antibodies. Therefore, the contention that IgA antibodies are irrelevant in the pathogenesis remains in doubt. IgA antibodies were not measured in the present study. However, our results show a lack of correlation between histopathology and IgG antibody titres as opposed to Czinn et al.⁵

In conclusion, this small pilot study failed to show a reasonable correlation between anti-*H. pylori* IgG and IgM titres and the severity of gastritis. Antibody titres do not appear to have a diagnostic and prognostic value. Histopathology remains the gold standard for the diagnosis of the disease.

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