Antibody titres to campylobacter pylori after treatment for gastritis

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Campylobacter pylori is strongly associated with histologically confirmed gastritis.[1,2] The most convincing evidence for a pathogenic role of *C pylori* in gastritis comes from trials of treatment.[3,4] We investigated the role of *C pylori* in dyspeptic patients who had gastritis confirmed by histology by performing a prospective trial with colloidal bismuth subcitrate 240 mg twice daily for four weeks. We also report the titres of antibody to *C pylori* before and after treatment.

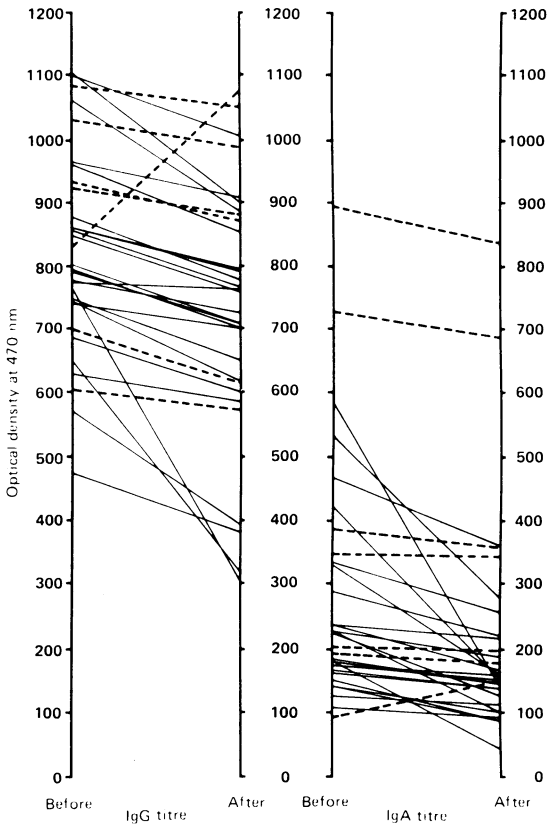
Patients, methods, and results

We studied 66 patients who had dyspepsia without ulcers and took three endoscopic biopsy specimens from both the gastric antrum and duodenal bulb. One specimen was placed in 10% buffered formalin for histology (colonising *C pylori* were identified by Giemsa stain), and the second was put in 6% urea solution (*C pylori* test).[5] The third was placed in 0·5 ml 20% sterile glucose, homogenised, treated with Gram stain, cultured on blood agar with amphotericin (6 μg/ml) and incubated at 37°C for six days. Patients who had a positive *C pylori* test result from the antrum started receiving treatment the same day as endoscopy. Blood was taken before and after treatment for antibody assessment by ELISA (enzyme linked immunosorbent assay) (sonicated antigen was used and titres determined at optical density 470 nm). Statistical analysis was by two tailed Mann-Whitney U test and matched paired *t* test after log transformation.

Antral histology showed acute or chronic gastritis, or both, in 32 patients, all of whom showed *C pylori* on microscopy. The 34 patients who had normal mucosal histology did not have *C pylori* and did not receive treatment. At the time of the second endoscopy (one week after finishing treatment) blood was taken, and careful questioning showed that only 19 of 31 patients had complied fully with the four week course of colloidal bismuth subcitrate, despite patients' and doctors' initial consent to participate in the study. Five patients received colloidal bismuth subcitrate for two weeks only, six ranitidine 300 mg daily for four weeks, and one metronidazole 200 mg every eight hours for two weeks. One patient was lost to follow up because he went overseas.

In 13 of 19 patients treated with colloidal bismuth subcitrate histology showed that gastritis had completely resolved and microscopy showed no *C pylori*. In six patients there was considerable improvement in the histological samples as well as a decrease in the amount of bacteria. Five patients who took colloidal bismuth subcitrate for only two weeks showed incomplete clearance of bacteria and gastritis. Histological samples from seven patients given ranitidine or metronidazole showed no change (four patients), mild improvement (one), or worsening (two).

Significantly higher IgG and IgA titres were found in patients who were positive for *C pylori* than in patients who were negative for *C pylori* ($p < 0{\cdot}001$; Mann-Whitney U test). IgG and IgA antibody titres were significantly decreased after colloidal bismuth subcitrate treatment (IgG=estimated change of ratios 1·21, confidence interval (CI) 1·10 to 1·32, $t=4{\cdot}31$, $p<0{\cdot}001$; IgA: estimated change 1·58, CI 1·33 to 1·86, $t=5{\cdot}57$, $p<0{\cdot}001$) whereas those in patients given ranitidine or metronidazole showed no significant difference (IgG: estimated change 1·02, CI 0·91 to 1·14, $t=0{\cdot}35$, $p>0{\cdot}1$; IgA: estimated change 0·98, CI 0·81 to 1·17, $t=0{\cdot}33$, $p>0{\cdot}1$). Indeed, some were even increased after treatment (figure). IgM titres were unchanged.



Serum IgG and IgA titres before and after treatment. ——=Colloidal bismuth subcitrate ($p<0{\cdot}001$). ---=Ranitidine and metronidazole (NS)

Comment

To our knowledge this is the first report of antibody titres before and after treatment. IgG and IgA antibody titres to *C pylori* may be useful in screening dyspeptic patients before endoscopy. The success of colloidal bismuth subcitrate and the inefficacy of H2-receptor antagonists in improving the histological features of gastritis as well as the number of *C pylori* is also confirmed. Increased titres of IgG and IgA to *C pylori* also provide evidence of active infection and may therefore be useful in following the response to treatment, compliance, and possibly reinfection.

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