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ABO blood group, secretor status and detection of *Helicobacter pylori* among patients with gastric or duodenal ulcers

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SUMMARY

Patients (454) referred for gastroscopy to the General Hospital of Athens were examined to determine (1) if non-secretors were over-represented among patients with ulcers and (2) is there was an association with ABO blood group or secretor status and carriage of *Helicobacter pylori*.

Compared with the local population, among patients with either gastric ulcer (51) or duodenal ulcer (96) there was a significant increase in the proportion of those who were blood group O ($P < 0{\cdot}025$); however, there were no significant differences in the proportions of non-secretors. *H. pylori* was identified in 62% of the 454 patients: 59·5% of those without evidence of ulcers; 62·5% of those with gastric ulcer; 88% of those with duodenal ulcer ($P < 0{\cdot}0005$). These bacteria were cultured more often and in higher numbers from patients with duodenal ulcer ($P < 0{\cdot}025$). There was no association between ABO blood group and prevalence of *H. pylori*. The prevalence of *H. pylori* among non-secretors with gastric ulcer (12·5%) was significantly lower than that for non-secretors with duodenal ulcer (100%) ($P < 0{\cdot}0005$). This was not observed for secretors.

INTRODUCTION

There are reports from the 1950s and 1960s that individuals of blood group O and those who are non-secretors of the glycoprotein form of their ABO blood group antigens are over-represented among patients with gastric or duodenal ulcers (reviewed by Mourant and colleagues) [1]. Non-secretion is associated with susceptibility to a number of infectious diseases (reviewed by Blackwell) [2, 3] and with asymptomatic carriage of pathogenic bacteria [4, 5] and yeasts [6, 7]. Non-secretors are also over-represented among patients with several autoimmune diseases for which infectious aetiologies have been postulated [4–11].

A recent review summarized the current evidence implicating *Helicobacter pylori* in the pathogenesis of gastroduodenal disease [12]. There is serological evidence

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that the majority (approximately 70%) of the adult Greek population (> 25 years of age) has been exposed to *H. pylori* [13]. In this study we examined ABO blood groups and secretor status (by means of Lewis phenotype) of patients referred for gastroscopy to determine: (1) if we could confirm the findings of earlier studies of associations between ABO phenotype and/or non-secretion and ulcers in a European population in which there is a high prevalence of *H. pylori*; and (2) to test the hypothesis that there might be an association with ABO blood group or secretor status and carriage of these bacteria.

SUBJECTS AND METHODS

The study population consisted of 454 consecutive patients between the ages of 17 and 86 referred for gastroscopy to the Gastroenterology Department of the General Hospital of Athens for investigation of symptoms of upper gastrointestinal disease. The majority of the patients had upper abdominal pain (58%), bleeding (melaena or haematemesis) (18%) or anaemia (11%). For men, anaemia was defined as red blood cell (RBC) count < 4500000/ml, haemoglobin (Hb) < 14 g/dl and haematocrit (HCT) < 42%. For women, anaemia was defined as RBC count < 4000000/ml, Hb < 12 g/dl and HCT < 37%.

For this study, during gastroscopy, three biopsy specimens were taken from the pylorus. Two specimens for culture were placed in separate containers with 1 ml of thioglycolate broth under aseptic conditions. A third specimen was fixed in 10% formalin, processed in paraffin and 3 μm sections were cut. The sections were stained with haematoxylin and eosin and examined by light microscopy for the presence of *H. pylori*.

Before culture, 0·8 ml of the broth was removed from the biopsy material and the specimen was vortexed in the remaining 0·2 ml with 4 or 5 glass beads. The material (0·1 ml) was then plated onto Skirrow medium composed of blood agar base, 7% horse blood, vancomycin (10 μg/ml), trimethoprim (5 μg/ml) and polymyxin B (2·5 IU/ml). Another 0·1 ml was plated onto medium containing Columbia blood agar base, 7% horse blood, and *Campylobacter pylori* supplement of vancomycin (10 μg/ml), trimethoprim (5 μg/ml), cefsulodin (5 μg/ml) and amphotericin B (5 μg/ml). The plates were incubated in microanaerobic conditions (Biomerieux system) for 7 days. The numbers of colonies per plate with characteristic appearance of *H. pylori* were counted and the numbers scored as follows: no growth = 0; 1–20 colonies = 1; 21–100 colonies = 2; 101–200 colonies = 3; > 200 colonies = 4. Colonies with characteristic appearance of *H. pylori* were examined by Gram stain and tested for production of oxidase, catalase and urease. A patient was classified as positive for *H. pylori* if the organism was identified either by culture or in the stained biopsy.

ABO blood group for each patient was determined by slide agglutination with monoclonal anti-A and anti-B antibodies (Scottish National Blood Transfusion Service) and the Lewis blood group phenotype by standard tube agglutination with monoclonal anti-Lewis^a and anti-Lewis^b antibodies (Scottish National Blood Transfusion Service). Individuals who are non-secretors can express only Lewis^a (Le^a) and those who are secretors can express Lewis ^{b} (Le^b). There is no association between sex, age and expression of ABO or Lewis phenotypes in adults. The

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results for the patient population were compared with those obtained with the same reagents for a series of 1248 individuals attending the analytical laboratory of the Hellenic Institute Pasteur. These were predominantly blood specimens taken for antenatal screening for antibodies to rubella and toxoplasma. The proportion of ABO phenotypes was similar to those reported previously for Greek populations [14, 15]. The proportions of Le^a and Le^b individuals in the patients attending the Institute were similar to the proportions of Le^a and Le^b individuals identified in a separate study of 891 male military results (Blackwell, unpublished results).

Epidemiological data, sex, age, symptoms, medication, smoking, alcohol consumption, results of biopsy reports, gastroscopy and culture results were coded and stored in a Data Base III plus data base. Results were analysed with the 'EXPLORE' statistical package, χ^2 test incorporating Yates' correction factor or student's t test.

RESULTS

Patients referred for gastroscopy

Of the 454 individuals (age range 17–86 years, mean age = 52 years) referred for gastroscopy, 60% were men (age range 18–86 years, mean age = 52 years) and 40% were women (age range 17–83 years, mean age = 51 years). There were 162 smokers (36%); 126 (78%) were men and 36 (22%) were women. The proportion of men who smoked was 126/270 (47%) and of women who smoked 36/181 (20%).

The proportion of individuals in whom *H. pylori* was identified by culture or by stained biopsy was 286 (68%). The proportions of men and of women in whom *H. pylori* was identified were 68 and 67% respectively. There were no significant associations when presence of the bacteria was analysed with reference to sex and smoking or sex and alcohol use.

The distribution of the ABO blood groups and Lewis phenotypes of the patient population is compared with that of the control population in Table 1.

Patients with ulcers

Results of gastroscopy identified 51 patients with gastric ulcer and 96 with duodenal ulcer. Among patients with gastric ulcer there were 31 men (61%) and 20 women (39%). Among the patients with duodenal ulcer, there were 78 men (81%) and 18 women (19%) which differed significantly compared with the total study population ($\chi^2 = 14{\cdot}9$, D.F. $= 1$, $P < 0{\cdot}0005$). For both men and women, the mean age was significantly higher among the patients with gastric ulcers compared to those with duodenal ulcers (Table 2).

Presence of ulcer was analysed by sex and smoking habits and by sex and alcohol consumption. No correlation was found with either of these factors.

Among patients with ulcers the proportion of those of blood group O was significantly increased compared with other blood groups (not O) (Table 3). Compared with the proportion of group O in the control population (40%), there were 29 patients of group O among the 51 patients with gastric ulcer (57%) ($\chi^2 = 5{\cdot}4$, D.F. $= 1$, $P < 0{\cdot}025$) and 51 of group O among the 96 patients with duodenal ulcer (53%) ($\chi^2 = 6{\cdot}2$, D.F. $= 1$, $P < 0{\cdot}025$). The proportion of patients with gastric

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Table 1. *ABO and Lewis phenotypes of patient population and controls*

ABO group	Patients No.	Patients (%)	Controls No.	Controls (%)
A	165	(36)	513	(41)
B	52	(11)	183	(15)
O	217	(48)	494	(40)
AB	20	(4)	58	(4)
Lewis phenotypes				
Le^{a+b-}	93	(21)	249	(20)
Le^{a-b+}	356	(78)	964	(77)
Le^{a-b-}	5	(1)	32	(3)

Table 2. *Ages of patients with gastric or duodenal ulcers*

Sex	Site of ulcer	Age range	Mean age
Men	Gastric	22–78	56*
	Duodenal	22–75	47
Women	Gastric	34–77	62†
	Duodenal	30–71	52

* $t = 2{\cdot}153$, D.F. = 105, $P < 0{\cdot}025$.
† $t = 2{\cdot}460$, D.F. = 36, $P < 0{\cdot}01$.

Table 3. *Distribution of ABO groups among patients with ulcers*

Blood group	Gastric ulcer ($n = 51$) %	Duodenal ulcer ($n = 96$) %	Control ($n = 1248$) %
A	31	35	41
B	10	10	15
O	57	53	40
AB	2	1	4

Gastric ulcer O/NOT O, $\chi^2 = 5{\cdot}4$, $P < 0{\cdot}025$
Duodenal ulcer O/NOT O, $\chi^2 = 6{\cdot}2$, $P < 0{\cdot}025$.

ulcer expressing Le^a was 16% and those expressing Le^b was 84%. Among those with duodenal ulcer, the proportion expressing Le^a was 27% and those expressing Le^b 73%. The figures for Lewis phenotypes did not differ significantly from those of the controls (Table 1).

There were 301 individuals in whom ulcers were not detected and for whom results of both culture and biopsy were available. *H. pylori* was identified in 179 (59·5%). The bacteria were identified in 30 of 48 (62·5%) patients with gastric ulcer for whom results of both culture and biopsy were available. It was identified among 82 of 93 (88%) patients with duodenal ulcer. The prevalence of the bacteria among the patients with duodenal ulcers was significantly higher than that of the study group ($\chi^2 = 19{\cdot}5$, D.F. = 1, $P < 0{\cdot}0005$).

Among the patients in whom no ulcer was found, *H. pylori* was identified in 46% of those who were group O and 54% of those who were not group O. *H. pylori* was identified in 64% of group O patients with gastric ulcer compared with 60%

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Table 4. *Quantitative isolation of* H. pylori *from patients without ulcers or gastric or duodenal ulcer*

Gastroscopy result	No growth	No. bacteria per biopsy 1–20	21–100	101–200	⩾ 200
No ulcer ($n = 308$)	153 (50%)	40 (13%)	36 (12%)	41 (13%)	38 (12%)
Gastric ulcer ($n = 48$)	22 (46%)	9 (19%)	6 (12%)	8 (17%)	3 (6%)
Duodenal ulcer ($n = 93$)	22 (24%)	20 (22%)	6 (6%)	23 (25%)	22 (24%)

No ulcer/duodenal ulcer $\chi^2 = 28{\cdot}37$, D.F. = 4, $P < 0{\cdot}0005$.
Gastric ulcer/duodenal ulcer $\chi^2 = 12{\cdot}81$, D.F. = 4, $P < 0{\cdot}025$.

among those who were not group O. The bacteria were identified among 92% of patients with duodenal ulcer who were group O compared with 88% of those who were not group O.

Among patients without ulcers, 34 of 59 non-secretors (58%) and 145 of 242 secretors (60%) were positive for the bacteria by either culture or biopsy. Analysis of the prevalence of *H. pylori* among non-secretors and secretors with gastric ulcer revealed significant differences; the bacteria were identified in only 1 of the 8 (12·5%) Le^a/non-secretors but in 28 of the 42 (67%) Le^b/secretors ($\chi^2 = 7{\cdot}5$, D.F. = 1, $P < 0{\cdot}01$). A similar analysis of the prevalence of *H. pylori* among non-secretors and secretors with duodenal ulcer revealed a different pattern; the bacteria were found in all the 24 non-secretors but in only 56 of the 67 (84%) secretors ($\chi^2 = 3{\cdot}1$, $P > 0{\cdot}05$).

Among patients who were non-secretors, the difference in the prevalence of the bacteria between those with gastric ulcer (12·5%) was significantly lower than that for this group with duodenal ulcer (100%) ($\chi^2 = 22{\cdot}0$, $P < 0{\cdot}0005$). Similar analysis of secretors did not find a significant difference in the prevalence of *H. pylori* among patients with gastric ulcer (67%) compared with those with duodenal ulcer (81%) ($\chi^2 = 1{\cdot}5$, $P > 0{\cdot}05$).

H. pylori was cultured more frequently from patients with duodenal ulcer (76%) than those with gastric ulcer (54%) or those without ulcers (50%). The proportions of those with 100 or more colony-forming units per biopsy specimen was 49% for those with duodenal ulcer compared with 23% for those with gastric ulcer ($\chi^2 = 12{\cdot}81$, D.F. = 4, $P < 0{\cdot}025$) of 25% for those without ulcers ($\chi^2 = 28{\cdot}37$, D.F. = 4, $P < 0{\cdot}0005$) (Table 4).

DISCUSSION

The present study confirms the earlier report from Athens [14] that individuals of blood group O are significantly over-represented among patients with either gastric or duodenal ulcers. The proportion of group O subjects in the population referred to the Institute Pasteur for analyses was 40% which is very similar to the figures reported by Hirszfeld and Hirszfeld (42%) in 1919 [15] and Merikas and colleagues (41%) in 1966 [14]. Compared with patients of the other blood groups

(not O), there was a significant increase in the proportion of group O individuals among patients with gastric (57%) or duodenal (53%) ulcers ($P < 0{\cdot}025$). In contrast to earlier findings for both British and Greek patients with ulcers [14, 16], there was not a significant association between group O and bleeding ulcers compared with non-bleeding ulcers. The number of patients in the present study was, however, smaller than those examined in the previous studies.

In this group of patients we were not able to confirm the reported associations for non-secretion and gastric ulcers (3/10 studies) or duodenal ulcers (13/15 studies (analysed by Mourant and colleagues) [1]. The proportion of Le^a/non-secretors in the population referred to the Institute for analyses was 20%, similar to that found in a separate study of 891 military recruits (21%) (Blackwell, unpublished results). The proportion of Le^a/non-secretors was 16% among patients with gastric ulcers and 27% among those with duodenal ulcers.

The original association of peptic ulcers with the presence of *H. pylori* has been confirmed by some investigators but not by others (reviewed by Buck) [12]. Although cigarette smoking has been identified as a risk factor for ulcers [17] and smoking is associated with carriage of some microorganisms [5], there was no association between smoking and presence of *H. pylori*. The proportion of patients with gastric ulcers in whom *H. pylori* was identified (62·5%) did not differ significantly from that of individuals in whom ulcers were not detected (59·5%) or the prevalence of antibodies to these bacteria in the general population of similar age. Among 458 blood donors (age range 21–50), 70% had serological evidence of exposure to *H. pylori* [13]. Among the patients in the study all but nine were 21 years of age or older and the proportion of males (60%) to females (40%) was the same as the blood donors. The prevalence of *H. pylori* among patients with duodenal ulcers (88%) was significantly increased compared with that of the study population ($P < 0{\cdot}0005$).

The results of this study suggest that in Greece where a high proportion of the population is exposed to *H. pylori* (1) there is not a significant association between the presence of these bacteria and gastric ulcers; (2) the numbers of *H. pylori* isolated from patients with gastric ulcers did not differ significantly from those isolated from patients without ulcers (Table 4). It has been reported that if other causes of gastric ulcer such as the use of the analgesics are excluded, the prevalence of *H. pylori* among patients with gastric ulcer approaches 100% [18]. In the present study only 9 patients were taking high doses of aspirin; 4 of these had gastric ulcers and *H. pylori* was isolated from 2.

The increased prevalence of *H. pylori* among patients with duodenal ulcers and the larger numbers of *H. pylori* isolated from these patients (Table 4) suggest the bacteria might contribute to development of the disease condition. These observations, the lower mean age of both men ($P < 0{\cdot}025$) and women ($P < 0{\cdot}01$) with duodenal ulcer (Table 2) and the lower proportion of women with duodenal ulcer ($P < 0{\cdot}0005$) suggest that a more detailed investigation of epidemiological factors influencing carriage of *H. pylori* in this population might be of value.

No association between presence of *H. pylori* and blood group O was found in patients without ulcers or those with ulcers. The proportion of non-secretors with gastric ulcers in whom the bacteria were found (12·5%) did not differ significantly from the proportion of non-secretors found in the group with gastric ulcers (16%);

but, the proportion of non-secretors with duodenal ulcers in whom bacteria were found (100%) was significantly increased compared with the proportion of non-secretors among those with duodenal ulcers (27%).

One of the hypotheses suggested to explain the increased susceptibility of non-secretors to particular diseases is that their mucosal surfaces might be more readily colonized by the causative microorganisms [2, 3]. Non-secretors are significantly over-represented among healthy asymptomatic carriers of *Streptococcus pyogenes* [4], *Neisseria meningitidis* [5] or *Candida albicans* [6, 7]. In the study by Burford-Mason and colleagues [6], non-secretion was associated with long-term carriage of the yeast. If *H. pylori* persists for longer periods or is present in larger numbers in the gastrointestinal tract, this might contribute to the pathogenic processes leading to ulcers. There were 24 patients in the present study who were re-examined by gastroscopy because symptoms had recurred or they had not responded to treatment. At the time of gastroscopy, none was being treated with bismuth and only one was taking an antibiotic. *H. pylori* was found in 4 of the 5 (80%) non-secretors but only 7 of the 19 (37%) secretors. The proportion of patients with > 100 colony forming units of *H. pylori* was significantly higher among the patients with duodenal ulcers; however, the numbers of patients were too small for statistical analysis by secretor status.

Several studies have reported that the epithelial cells of non-secretors bind larger numbers of some microorganisms than cells of secretors: uropathogenic strains of *Escherichia coli* [19]; *C. albicans* [20, 21]; and meningococci [unpublished results]. Although bacteria can bind to proteins on epithelial surfaces, carbohydrates appear to be the receptors recognized by many bacterial adhesins, probably due to their abundance and variety [22]. There is evidence that blood group antigens act as receptors for several microorganisms. The P blood group antigen is a receptor for some uropathogenic strains of *E. coli* [23]; and the Duffy blood group antigen acts as a receptor for the malaria parasite *Plasmodium knowlesi* [24].

Two blood group antigens common to most individuals have been proposed to act as receptors for microorganisms, H and Le^a [2, 3]. H, the antigen of blood group O, is found on the cells of all individuals except the very rare Bombay phenotype [25]. The Lewis antigens on epithelial cells are adsorbed from secretions and reflect those present in the body fluids. Although secretors express Le^b predominantly, some can have substantial amounts of Le^a in their body fluids, and consequently on their epithelial cells [26, 27]. The amount of Le^a present in secretors depends on the efficiency of the fucosyl transferase coded for by the secretor gene [28].

Previous studies by our group have found that buccal epithelial cells of secretors express 3–6 times more H than those of non-secretors [29]. If H were one of the receptors for microorganisms, there should be increased attachment to cells of secretors. A second piece of evidence suggests H is not a receptor for *H. pylori*. A gastric glycolipid to which *H. pylori* binds is also found on red blood cells of groups, A, B and O; however, this substance is not one of the ABO blood group glycolipids [30].

There is evidence that glycoproteins containing sialic acids in their sugar moieties can inhibit binding of *H. pylori* to erythrocytes. The carbohydrates are

suggested to bind a fibrillar haemagglutinin [31]. The inhibitory substances are found in porcine gastric mucin, to a lesser extent in bovine submaxillary mucin [32] and in human saliva [33]. Neuraminidase treatment of human saliva reduced its ability to inhibit haemagglutination. This indicated that sialic residues contribute to, but are not totally responsible for, the inhibitory effect of the salivary mucin [33]. The role of Lewis antigens in these interactions is under investigation.

At present little is known about the distribution or amounts of the Le^a antigen on epithelial cells in the stomach and duodenum. Although non-secretors cannot produce Le^b, secretors can have varying amounts of Le^a in body fluids. Quantitative differences in the amount of Le^a present in mucus of secretors and non-secretors of patients with ulcers compared to those without ulcers have not been determined. These and other studies are currently underway to test the hypothesis that Le^a might be one of the receptors for *H. pylori* in the gastrointestinal tract.

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