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Helicobacter pylori and ABO blood groups

R J L F Loffeld, E Stobberingh

Abstract

A serological study was carried out to assess the prevalence of antibodies to *Helicobacter pylori* and compare it with the distribution of ABO blood groups. Serum samples from 402 healthy blood donors were tested with an IgG enzyme linked immunosorbent assay. There was no difference in blood groups between those who were seropositive and those who were seronegative, which suggests that blood group O is not a risk factor for acquiring *H pylori* infection.

Helicobacter pylori has been implicated as the major cause of type B (antral) gastritis. The bacterium also has an important role in the pathogenesis of duodenal ulcer disease.¹⁻³ Blood group O has been regarded as a risk factor for duodenal ulcers for many years,⁴ although the reasons for this are not clear.

Because duodenal ulcer disease is associated with antral *H pylori* infection in 90-100% of the cases, blood group O might also be a risk factor for acquiring *H pylori* infection.

A prospective study was done in a group of healthy subjects. The presence of IgG antibodies against *H pylori* was compared in ABO blood groups with the prevalence of Rhesus factor.

Methods

The group comprised 402 healthy blood donors (363 men and 39 women, mean age 42 years, range 19-65 years). ABO blood group

and rhesus factor were determined with standard serological tests (Centraal laboratorium van de Bloedtransfusiedienst, CLB, Amsterdam, The Netherlands). Sera were taken for the assessment of IgG antibodies against *H pylori* with an enzyme linked immunosorbent assay (ELISA), using a crude sonicate of five different strains of *H pylori* as antigen. This assay has sensitivity and specificity values of 98% and 94%, respectively. This method has been described previously.⁵

Statistical analysis was done with the χ^2 test.

Results

One hundred and forty three (35.5%) subjects had antibodies against *H pylori*, the remainder (259 or 64.5%) were seronegative. Of those seropositive, 112 (78.3%) were rhesus D positive compared with 219 (84.5%) of those who were seronegative. This difference was not significant.

One hundred and seventy six (43.8%) blood donors were of blood group O; 179 (44.5%) were of blood group A. Blood groups B and AB occurred in 34 (8.4%) and 13 (3.3%) subjects, respectively. The table shows the correlation between blood groups and the presence of *H pylori* antibodies. There was no significant difference in blood group distribution among seropositive and seronegative subjects.

Discussion

In a recent report no association was found between blood group, antibody prevalence, and *H pylori* infection.⁶ In this study *H pylori* was detected using a rapid urease method for gastric biopsy specimens. This could be a possible drawback, because an unknown number of patients could have cleared or eradicated *H pylori* as a result of concomitant treatment with antibiotics or bismuth preparations. Serological studies for

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Blood groups and IgG antibodies in healthy subjects

	Blood group				Total numbers
	O	A	B	AB	
Seropositive	56 (39%)	70 (49%)	13 (9%)	4 (3%)	143
Seronegative	120 (46%)	109 (42%)	21 (8%)	9 (4%)	259

antibodies against *H pylori* are indirect methods for detection of the micro-organism. Seropositivity, therefore, can reflect active infection as well as (especially if the antibody titres are low) previous infection (a so-called serological scar). Although this phenomenon can interfere with the diagnosis of acute *Helicobacter* infection, it is of no relevance in our study. Seropositivity thus indicates present or previous infection with *H pylori*. Because blood group is genetically determined, this does not interfere with the aim of the study, nor influence the final results.

We confirm the data from the previous biopsy study. Our data also agree with those of a smaller serological study published recently.⁷

It is concluded that although blood group O is a risk factor for duodenal ulcers, it is not a risk factor for acquiring *H pylori* infection.

Blood group O and *H pylori* are independent risk factors for duodenal ulcer disease.

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Anticardiolipin antibodies in leptospirosis

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Abstract

The clinical course and serology of 16 cases of leptospirosis in an area with an unusually high endemic infection rate were studied to gain further insight into the pathology of the secondary immune phase that is typical of the disease. IgG anticardiolipin antibody concentrations were measured by immunoassay and found to be increased in eight serologically confirmed cases with severe complicated disease, compared with eight patients with relatively uncomplicated leptospirosis who had IgG anticardiolipin concentrations within the control reference range.

This previously unreported association suggests that leptospira may induce vascular endothelial injury in severe cases and expose crypt antigens or induce conformational change of cell surface phospholipids. Leptospirosis may provide a model for an infective origin of some cases of the antiphospholipid syndrome.

Syphilis can induce the cross-reaction of

antibodies with synthetic cardiolipin. More recently, IgG and IgM anticardiolipin antibodies have been reported in patients with Lyme disease, another spirochaetal disease.¹ The antigenic stimulus responsible for the induction of these antibodies in spirochaetal infection, and their relation with the complications of the disease is poorly understood.

Leptospirosis is a spirochaetal infection with important animal reservoirs in rodents, dogs, cattle and pigs. The initial septicaemic phase of the infection is characterised by fever and malaise. Although severe cases may progress to hepato-renal disease (Weil's syndrome), most are anicteric, and without confirmatory serology, may remain undiagnosed.² The second or "immune phase" occurs about two weeks later and is associated with a rise in circulating IgM leptospira antibodies and recurrence of fever. Encephalitis, neuritis, thrombocytopenia, heart-block and cardiac failure may manifest at this time, suggesting that an immunological mechanism rather than a direct cytopathic effect may be responsible for these complications.^{3,4}

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