Blood group, secretor status, and susceptibility to infection by *Neisseria gonorrhoeae*

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SUMMARY To determine whether the presence or absence of anti-B isohaemagglutinin in individuals of blood group B increases their susceptibility to gonococcal infections 567 new patients attending a sexually transmitted disease clinic were screened for blood group and secretor status. Of the patients with blood group B, 20·1% had gonorrhoea and 12% had not. A higher percentage (20·9%) of patients with no anti-B isohaemagglutinin had gonorrhoea compared with those without (12·1%). There was, however, no synergy between the absence of anti-B isohaemagglutinin and non-secretion of water-soluble blood group B antigen. Further research is needed to determine the underlying host-parasite interactions responsible for the increased susceptibility to gonorrhoea in these individuals.

Introduction

Over the past six years there have been reports of increased susceptibility of individuals of blood group B to infection with *Neisseria gonorrhoeae*.^{1,2} Foster and Labrum¹ suggested that the presence or absence of anti-B isohaemagglutinin may be the causal factor for the reported increase in susceptibility to gonorrhoea of individuals with blood group B. In a recent study we found that individuals of blood groups B and AB — that is, those with no anti-B isohaemagglutinin — who were also non-secretors of blood group antigen were significantly more susceptible to urinary tract infection.³ To determine whether there is a synergistic effect between those two host factors and susceptibility to gonococcal infection we screened patients attending a department of genitourinary medicine for blood group and secretor status, and we related the data to the occurrence of gonorrhoea in these patients.

Patients and methods

During a three-month period all new patients attending the department of genitourinary medicine at the Royal Infirmary, Edinburgh, were asked to provide a specimen of blood and saliva for determination of blood group and secretor status.

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Control data for blood group frequency were available for 6662 donors from the same geographical area.⁴ Secretor status control data were produced by testing 334 plasma specimens from blood donors in the survey area.

SCREENING PROCEDURES

Blood from each patient was collected at the clinic and stored in heparinised tubes. The saliva from each patient was boiled for 20 minutes, centrifuged at $500 \times g$ for 10 minutes and the supernate stored briefly at 4°C until tested for the presence of blood group antigen.

Blood group was determined by agglutination tests in plastic wells (WHO plates). Secretor status was determined as described by Mollinson⁵ with saliva or plasma samples. Samples of saliva or plasma from secretors and non-secretors representative of the four blood groups were used as controls for each experiment. Agglutinins used were anti-A and anti-B sera and *Ulex europaeus* lectin. The red blood cells used were of groups A₁, B, and O. The Blood Transfusion Service, Royal Infirmary, Edinburgh, kindly supplied the reagents for blood grouping and determination of secretor status and also performed random verification tests on 50 selected saliva samples and 46 plasma samples.

Results

The distribution of blood groups of patients with and without gonorrhoea and of the controls is given in

TABLE I Distribution of ABO blood groups in 567 patients with and without gonorrhoea and controls

Blood group	Culture results for <i>N gonorrhoeae</i>		Total No (%) of patients	No (%) of controls ⁴
	No (%) negative	No (%) positive		
O	247(50.3)	35(46.1)	282(49.7)	3323(49.9)
A	176(35.8)	23(30.3)	199(35.1)	2410(36.2)
B	59(12.0)	16(21.1)	75(13.2)	715(10.7)
AB	9(1.8)	2(2.6)	11(1.9)	214(3.2)
Total	491(99.9)	76(100.1)	567(99.9)	6662(100.0)

table I. The blood group frequencies of the controls and the total number of patients attending the clinic showed no significant differences ($\chi^2 = 5.864$, $p > 0.1$). The frequency of blood group B in these patients with gonorrhoea was 21.1% compared with 12% in those without. This difference was not, however, significant ($\chi^2 = 5.103$, $p > 0.1$). When the frequency of blood group B in the patients with gonorrhoea was compared (21.1%) with that of the controls (10.7%) the difference ($\chi^2 = 8.404$, $p < 0.05$) was significant.

The effect of the presence or absence of anti-B on susceptibility to gonorrhoea is shown in table II. A higher percentage (20.9%) of patients with no anti-B isohaemagglutinin had gonorrhoea compared with those with anti-B isohaemagglutinin (12.1%) ($\chi^2 = 4.947$, $p < 0.05$). Using the relative risk method of Woolf⁶ we found that the relative risk of gonorrhoea was 1.93 for individuals without anti-B isohaemagglutinin. This means that they are 93% more susceptible to gonorrhoea than persons with anti-B isohaemagglutinin.

The distribution of secretor status for patients with and without gonorrhoea and controls is given in table III. No significant differences were noted between the total patients and the controls ($\chi^2 = 0.917$, $p > 0.1$) nor between those patients with and without gonorrhoea ($\chi^2 = 0.168$, $p > 0.5$).

TABLE II Analysis of data from table I by presence or absence of anti-B isohaemagglutinin

Anti-B	Culture results for <i>N gonorrhoeae</i>		Total
	No (%) negative	No (%) positive	
Present (O and A)	423(87.9)	58(12.1)	481(100)
Absent (B and AB)	68(79.1)	18(20.9)	86(100)
Total	491	76	567

TABLE III Distribution of secretor status for patients with and without gonorrhoea and controls

Secretor status	Culture results for <i>N gonorrhoeae</i>		Total No (%) of patients	No (%) of controls
	No (%) negative	No (%) positive		
Secretor	344(70.1)	55(72.4)	399(70.4)	245(73.4)
Non-secretor	147(29.9)	21(27.6)	168(29.6)	89(26.6)
Total	491(100)	76(100)	567(100)	334(100)

Discussion

Our findings confirm those of others^{1,2} that individuals of blood group B are more susceptible to gonococcal infection and further suggest that the absence of anti-B isohaemagglutinin is important.

An increase in the number of non-secretors among patients with gonococcal infections had been predicted, but this was not observed. Although there was a significant increase in the number of blood group B individuals among the infected patients, there was no synergy between the absence of anti-B isohaemagglutinin and non-secretion of water soluble blood group B antigen comparable to that found in patients with urinary tract infections.³

The underlying host-parasite interactions responsible for the increased susceptibility of individuals with no anti-B isohaemagglutinin to gonococcal infection are not yet known; they appear, however, to differ from those involved with other Gram-negative, urinary tract pathogens. Accordingly, we are currently investigating the role of anti-B isohaemagglutinin in normal human serum acting as an opsonin or as bactericidal antibody, gonococcal interactions with human phagocytic cells of the different blood groups, and differences in the attachment of gonococcal strains to epithelial cells from individuals with different ABO blood groups.

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