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ABH Secretor Status in Patients with Bladder Tumours

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Summary. The ABO blood groups and secretor status of a group of patients with bladder tumours were investigated and compared with those of a group of patients of a similar age suffering from a variety of genito-urinary diseases. Some of the control patients were known to have been exposed to potential carcinogens, and the findings in this group were analysed separately. No significant differences were found when comparisons of these three groups of patients were made.

Key words: Bladder tumours - ABH secretor status - Genetics.

There have been few studies on the relationship between blood groups and genito-urinary disease. Pesce (11), Vesely (13) and Charzewski (3) investigated the relationship between stone disease and blood groups. In a similar study Anand (2) made brief reference to secretor status but did not discuss this aspect further. Okajima and Hiramatsu (9) investigated the ABO blood groups of patients with bladder tumours, without mention of secretor status, and did not find any significant correlation.

The present study was undertaken to decide whether or not the bladder epithelium of subjects of various ABO and secretor types is particularly prone to develop tumours.

PATIENTS

Studies were made on 85 male patients. There were 30 patients with bladder tumours (Table 1) and fifty-five patients with a variety of urological conditions who were similarly investigated as a control group. (Table 2). All patients were asked to supply a full occupational history since leaving school and to give details of their past and present smoking habits. A cigarette smoker was arbitarily defined as someone who had smoked more than five pack years[1]. Patients with overt respiratory infection were excluded.

1 A "pack year" = 20 cigarettes per day for one year, or the equvalent eg. 40 cigarettes per day for 6 months.

Male patients only were studied as many females found it difficult or distasteful to produce the requisite sample of saliva.

METHOD

Patients were asked to provide at least 5 mls of saliva, without the use of a sialogogue. The blood group of the patient was recorded.

The sample of saliva was heated in a boiling water-bath for ten minutes in order to inactivate salivary enzymes, and then centrifuged at 5,000 r. p. m. for one hour to remove food debris. The supernatant was either tested immediately or stored at -4° C.

The presence or absence of H substance in the saliva was determined using a purified extract of Ulex Europaeus seed (Lectin-H, Dade Division, American Hospital Supply Corporation). One drop of the prepared saliva was incubated at room temperature for 10 min with one drop of a 1 in 6 dilution of Lectin-H in 0.85 % saline. One drop of a freshly prepared 2 % suspension of once-washed group O cells in physiological saline was then added, and the mixture incubated at room temperature for a further 5 min.

The resulting suspension was then centrifuged at 1,000 r. p. m. for one minute and observed macroscopically for agglutination.

When saliva contains H substance there is an inhibition of Lectin-H, which is therefore unable to agglutinate the red cells. If, on the other hand, the subject is a non-secretor, agglutination may be observed.

Table 1. Patients with bladder tumours

Histological type	Number	Mean age at presentation	Exposure to a known carcinogen Number	Nature
Transitional cell carcinoma	28	62	16	All cigarette smokers, including one man who had worked as a vulcaniser
Squamous cell carcinoma	2	34		None
Total	30	51	16	

Table 2. Control patients

Diagnosis	Number	Mean age	Exposure to a known carcinogen Number	Nature
Benign enlargement of the prostate	17	63.3	6	All cigarette smokers
Stone disease	9	41.3	3	All cigarette smokers
Infertility (tubular 'sloughing')	4	25.7	1	Cigarette smoker
Carcinoma of the prostate	3	61.3	1	Cigarette smoker
Urethral stricture	3	46.6	2	One cigarette smoker One had worked as a maintainence electrician in a dyestuffs factory
Miscellaneous group	19	44.0	9	All cigarette smokers
Total	55	49.3	22	

The miscellaneous group consists of patients with epididymal cysts (2), hydronephrosis (2), hydrocele (2), seminoma (2), not yet diagnosed (2), bladder neck dyskinesis (2), phimosis (1), benign solitory renal cyst (1), carcinoma of the kidney (1), torsion of the testis (1), urethritis (1) and varicocele (1) and one man wishing to have a vasectomy.

RESULTS

The 55 control patients (Group 1) showed a 73% prevalence of positive secretor status, with 44% blood group A, 12% group B, 4% group AB and 40% group O. These figures are similar to the distribution of ABO groups in the West of Scotland:

40% A, 12% B, 3% AB, 45% O (figures supplied by the Blood Transfusion service). Although there are no comparable figures for secretor status in this region, a study of 2,435 subjects in Liverpool - recognised as not being strictly comparable from a genetic viewpoint - showed a 76% predominance of positive secretor status (10).

As the distribution of ABO genes in the population at large and in the control group is so similar it may be assumed that the controls are a representative sample, and presumably their distribution of secretor genes does not differ greatly from the norm.

The 22 controls who had been exposed to a possible carcinogen (Group 2) showed a distribution of ABO and secretor genes which was not significantly different from the total control group. Group 2 had a 68% prevalence of positive secretor status, 36% were group A, 14% group B, 5% group AB and 45% group O.

The patients with bladder tumours (group 3) were 63% secretors, 33% group A, 16% group B, 7% group AB and 43% group O.

Table 3

ABO groups and secretor status in control group (group 1)

	A	B	AB	O	Total
Secretor	17 (31%)	5 (9%)	1 (2%)	17 (31%)	40 (73%)
Non-secretor	7 (13%)	2 (3%)	1 (2%)	5 (9%)	15 (27%)

ABO groups and secretor status in those controls exposed to carinogen (group 2)

	A	B	AB	O	Total
Secretor	6 (27%)	3 (14%)	0	6 (27%)	15 (68%)
Non-secretor	2 (9%)	0	1 (5%)	4 (18%)	7 (32%)

ABO groups and secretor status in patients with bladder tumours (group 3)

	A	B	AB	O	Total
Secretor	7 (23%)	4 (13%)	2 (7%)	6 (20%)	19 (63%)
Non-secretor	3 (10%)	1 (3%)	0	7 (23%)	11 (37%)

When the results in groups 2 and 3 are compared the distribution of blood groups and secretor types is virtually identical (Yates $X^2 = 0.0035$). Neither is there any significant difference between groups 1 and 3.

DISCUSSION

The blood group specific mucopolysaccharides are secreted in all body fluids of secretors and the concentration is highest in saliva, which is therefore taken for routine analysis (12). A reason for not making a direct study of these substances in urine is that certain pathogens, notably E. Coli, Proteus and Pseudomonas can bear antigens indistinguishable from the blood group substances (4). Similarly, patients whose sputum contained these organisms could give a false positive result - and therefore patients with overt respiratory disease were excluded from this study.

There is an association between blood group O and non-secretion and duodenal ulcer (10) and it has been postulated that the mucopolysaccharide blood group substances may have a direct protective effect on the duodenal mucosa, H substance being less protective against hydrochloric acid than either A substance or B. substance (1). The relationship could also be explained by stimulation of the gastric cell mass by H substance (6). There are definite differences in the behaviour of established duodenal ulcer dependant on the patient's ABO group and secretor status: non-secretors requiring surgery for peptic ulcer more frequently than secretors with the same disease (8) and there is a stronger association between blood group O and those ulcers which bled than with those which did not (5).

Another interesting facet of the relationship between blood groups and secretor status and gastro-intestinal pathology lies in the different responses of the gastric mucosa of patients of these genetic types to the exogenous trauma of aspirin wash-outs: more cells are shed, in response to this insult, from the stomachs of O secretors than O non-secretors and from those of A non-secretors than of A secretors (7).

Many factors are involved, and McConnell concluded "the correct explanation may depend partly on Aird's protection theory and partly on Hanley's acid stimulation theory" (10).

If Aird's protection theory is tenable, it is possible that group specific mucopolysaccharide might protect other epithelial surfaces against other forms of chemical trauma. The present study was designed to test this hypothesis with reference to bladder tumours and exogenous carcinogens. If, indeed, the blood group mucopolysaccharides in the urine of secretors were protective one would expect those patients who had been exposed to carcinogens and did not develop bladder tumours to show a preponderance of secretors. Conversely, the bladder tumour group might be expected to contain few secretors. Similarly, if there was a genetic predisposition to bladder tumours associated with a particular blood group/secretor

type, then this would be obvious from the distribution in the group 3 subjects.

The results of this study do not support these speculations. Blood group mucopolysaccharide in the urine does not appear to offer protection against chemical carcinogens as shown by the virtually identical distribution of secretors and non-secretors in control patients exposed to a carcinogen compared to patients with proven bladder cancer. It has also been shown that none of the genetic types studied have a predisposition to bladder tumours.

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