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NON SECRETION OF BLOOD GROUP ANTIGENS AND SUSCEPTIBILITY TO URINARY LRACT INFECTION

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SUMMARY

The material of the present study comprised 103 patients of urinary tract infection confirmed by bacterial examination of urine, (64) out of them had chronic renal failure, due to recurrent urinary tract infection. These patients underwent determination of blood groups (ABO and Lewis) and secretor status, to find out if there is relation between it and susceptibility to urinary tract infection. There was a predominence of blood group AB and non secretor status (Se Se) which proves that the absence of A & B antibodies and also absence of Se gene combine to give an increased risk to urinary tract infection. The appearance of increased frequency of Lea (a-b) in non

secretors confirms the consumption of Le antigen as receptors for pathogenic organisms. There is no predominence of special blood groups in patients not confirmed to be recurrent.

INTRODUCTION

The antigenic determinants of the ABO and Lewis systems in addition to red cells also exist in the body secretions in soluble form when the relevant genes are expressed in the phenotype. The antigens expressed on both red cells and in the secretions are determined by the interaction of Hh, Sese ABO and Le le genes (Green, 1989).

One of the innate defences against MANSOURA MEDICAL JOURNAL

superficial infection appears to be the ability of an individual to secrete the water soluble form of ABO (Thom et al., 1989).

Blackwell and Weir (1989) suggested that Lewis of non secretors might be one of the receptors for some yeast strains (Candida Blastospores). It is postulated that blood group substances may interfere with the adherence of streptococcus mutans to teet (Halbroak & Blackwell, 1989); Shinebaum (1989) proved that there is significantly increase of prevalance of non secoretors amongst different groups of patients with spondyloarthropathy, ankylosing spondylitis, reactive arthritis and Psoriatic arthropathy.

The excretion of blood group substances in urine is an active process in the kidney and is not a simple filtration (Kalinowski, 1972). These substances may have a protective part to play in secretions. Blakwell et al., (1986) found a significant increased prevalence of non secretors of ABH blood group antigens into body fluids

among patients with recurrent urinary tract infection or superficial infection with Candida albicans. Lomberg et al. (1986) found that the degree of attachement of E. Coli to uroepithelial cells from non secretors was higher than that to cells from secretors. Drack et al., (1971) found A and B blood group activity in 47% of 34 urinary tract pathogens. Kinane et al. (1982) found that individuals of blood group AB with no anti-B isohaemagglutinins and non secretors were more susceptible to recurrent urinary trat infection.

Possible host-parasite interactions underlying the increased proportion of non secretors among women with recurrent urinary tract infections and those leading to development of renal scars are discussed by May et al. (1989) and in their study they suggegted that non secretion might influsence the Pathogenic sequelae of these infections. The strains of E. Coli isolated from urine of non secretors are more powerful in virulence and production of haemolysin than those isolated from secretors (May et al.,

1989b).

The aim of this study is to determine the host factors of ABO & Lewis blood groups and secretor status and to confirm the synergistic associations of these factors with susceptibility to urinary tract infection and chronic renal failure.

MATERIAL AND METHODS * Patients:

103 patients with age ranged from 25-48 were examined of them:

- a) 42 having pyuria, 25 males and 17 females.
- b) 64 complaining of chronic renal failure with history of previous recurrent urinary tract infection.
 They were on maintenance haemodialysis preparing them for renal kidney transplantation.

Control:

125 normal persons of matched age and sex were selected for blood grouping and determination of secretor.

* Methods :

Bacterial examination were done to 42 cases by collecting mid-stream specimen of urine (MSU) in sterile container. The samples were cultured on nutrient agar, blood agar and Mac ConKey. The organigms are identified by gram stained film, pathogenic staphylococci were confirmed by coagulase test and identification of gram negative bacilli were performed by biochemical reaction using enterotube 11 Roche.

A specimen of blood was taken from each case on sterilized tube for blood grouping (ABO and Lewis), ABO were determined by agglutination tests using anti-A, anti-B, anti-AB (O serum), anti-A to determine, A or A B and anti-H for A2 or A2B. Thege groupg were confirmed by reverse grouping using the gerum againgt Al, B and O cellg (Simmong, 1980).

Lewis group were determined by saline agglutination in tube using anti-Le ^a for Le^a and anti-Le^a for Le^a after incubation 1/2 hour in room temperature for Le^a and 4 c for Le^a (Dacie &

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Lewis, 1984).

For determination of secretor status, we prefere the saliva as it is the most easy and available and the urine is not available as it contains many enzyme which may lead to deterioration of group substances. Saliva (2.5 ml) were collected from each individual for determination of secretor status by haemaggglutimation inhibition test (neutralization test) using anti- A, anti-B and anti-H against A,B and O cells (Simmong, 1980).

RESULTS

ABO, secretor status and Lewis distribution in patients compared to control are shown in table (1-5)

DISCUSSION

Secretion of blood group antigens appears to play a part in the innnate defences at the mucosal surface (Blackwell et al., 1986).

In this study, there was significant predominence of blood group AB as shown in table 1 (x2 of difference 6.32). This proves that the absence of

corresponding isohaemagglutinins increases the susceptibility to recurrent urinary tract infections. This suggests that isohaemaggutinins may interact with blood group like antigens on bacterial cell walls to inhibit attachement to uroepithelial cells and when complement was added a bacteriocidal or bacteriologic reaction ensued. This was in accordance with what was reported by Kinane et al. (1982). While in table (4), it was found that there is no predominance of special blood group either in staphylococcus cr gram negative bacilli (E. Coli), -in patients with recurrent urinary tract infection.

From table (2 and 5), it was found that the non secretor status was significantly more frequent in patients than control (X 20f difference: 14.94 in table 2 and 14.8 in table 5). This proves that the secretion of blood group substances, in either staphylococcus or gram negative bacilli infection, has a protective role by either ability to occupy or in some way they interfere with binding sites either on the bacterium or on the epithelial cells

with possible effects on bacterial colonization and subsequent invasion and infection. This was in accordance with the results of Weir et al. (1981), Blackwell et al. (1986), Lomberg et al. (1986) and Thom et al. (1989).

In table (3) the significantly increased frequency of Le^a (a-b) Prove that Le^a antigen in non secretor has a role in infection as it appears to be consumed as receptors to bacterium and then this antigen binds after wards to the epithelial surfaces by another portion of it. This explanation is in agreement with Blackwell et al. (1986). This was also Proved by

Lomberg et al. (1986) who found that the attachement of bacterium to uroepithelial cells was higher in non-secretors and there is a shielding of receptors (Le antigen) by products controlled by secretor genes. This is in agreement with our results which suggest the shielding and disappearance of Lea antigen in non secretor genes (X2 for Le^a (a-b-) / Le^a (a + b-): 29.53).

We concluded that secretion of blood group and secretory state may provide additional information in identifying those at risk.

Table (1): Frequency of ABO blood groups, the relative incidence and values in urinary tract injection.

	-						
Blood							
	No.	%	No.	%		RI	x ²
Al			39	200000 1100000	7.7.7.7.		
A2	34/37	35.92	7	32.2	A/D	1.21	0.35
А3			2	7.2			
	21	20.39	25	20	B/D	1.32	0.53
A1	11						
A2	5 17	16.50	8	6.4	A/D	3.43	6.32
АЗ	1						
	28	27.18	44	35.2	Non O / O	1.46	1.68
	A2 A3 A1 A2	Al A2 34/37 A3 21 A1 11 A2 5 17 A3 1	Al A2 34/37 35.92 A3 21 20.39 A1 11 A2 5 17 16.50 A3 1	Al 39 A2 34/37 35.92 7 A3 2 21 20.39 25 A1 11 A2 5 17 16.50 8 A3 1	No. % No. % AI 39 A2 34/37 35.92 7 32.2 A3 2 7.2 21 20.39 25 20 A1 11 A2 5 17 16.50 8 6.4 A3 1	Al 39 A2 34/37 35.92 7 32.2 A/D A3 21 20.39 25 20 B/D A1 11 A2 5 17 16.50 8 6.4 A/D A3 1	No. % No. % RI AI 39 A2 34/37 35.92 7 32.2 A/D 1.21 A3 2 7.2 21 20.39 25 20 B/D 1.32 A1 11 A2 5 17 16.50 8 6.4 A/D 3.43 A3 1

^{\$} significant

Table (2): The phenotye, and gene fraquenies of secretor and non - secretor, the relative index and X2 urinary tract infection

			Secretors		Non secretors	Sec / non SErcr .	
	Total.	No .	Se Gene Freq.	No .	Be Gene Freq.	RI	x ²
Control group	125	95	0.51	30	0.49		
		(67%)	(51%)	(24%)	(49%)		
Patients group	103	53	0.3038	50	0.6967	0.33	14.94
		(51.4%)	(30.33%)	(48.54%)	(69.6%)		

^{\$} significant

Table 3: Distribution of Lewis blood groups in urinary tract infection and control .

Secretor Status	Lewis system	Lewis system (125)		Patients (103)		12 Julian score	
		No .	%.	No .	%.	x²	
Secretors	Le(a-b+)	84	67.2	48	46.6	Le(a-B-)/Le(a-b+)	
	Le(a-b-)	11	3.8	5	4.8	0.162	
Non secretors	Le(a+b-)	26	20.8	38	36.89	Le(a-b-)/Le(a+b-)	
	Le(a-b-)	4	3.2	12	11.65	29.53@	

[@] significant

Table 4: Frequency of ABO blood groups and the relative incidence and X 2 values in both Staphylococcus and E. Coli urinary, tract infection.

Blood group	E , coli	Staphylococci	Total No	Control	Compared group	RI	x ²
A1 A	5 6 1	6	12	48	A /O	0.65	1.03
3	6	5	11	25	B/O	1.14	0.08
AB	1	1	2	8	AB/O	0.65	0.27
	10	7	17	44	non O / O	0.9	0.06

Table 5: Frequency of ABO blood groups and the relative incidence and X 2 values in both Staphylococcus and E. Coli urinary, tract infection.

Group	Total	Secretors		Sec / non sectr		
specificity	No.	Secisions	Non secretors	R.1	x ²	
Control group Urinary tract Infection	125	95	30			
E . coli	23	12	11	1.9	5.51@	
Staphylococci	19	8	11	0.72	9.30@	
Total	42	20	22		14.81@	

[@] significant

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