Lewis Blood Groups in Patients With Peptic Ulcer Disease: Association With Secretor Status

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Aims and objective of the study is to find Lewis blood group association with secretor status in peptic ulcer disease patients. Lewis blood grouping was done by direct agglutination test and secretor status by agglutination inhibition test. 50 controls and 50 endoscopically diagnosed PUD patients were selected from different hospitals of Lahore. Controls were taken from blood banks. Duodenal ulcer is not associated with any Lewis phenotype or secretor status.

Key words: Lewis, secretor status

Lewis antigens are produced by tissue cells, secreted into body fluids and then absorbed onto the red cell membrane from plasma. The main Lewis antigens Le^a and Le^b, result from the interaction of the negative or positive alleles at Lewis and secretor gene loci. The Le gene is inherited independent of ABO, Hh and Sese genes. The possible Lewis phenotypes of adult red cells are Le (a+b-), Le (a-b+), Le (a-b-) and Le (a+b+). The Lewis genes control the production of specific transferase enzymes that cause the addition of a single sugar residue to a preformed precursor substance which is same for ABH and Le gene. The difference in the terminal sugar determines these antigens. Anti-Le^a and anti-Le^b are main Lewis antibodies[1,2].

The individuals who secrete the water soluble A, B and H substances in saliva and other body fluids like gastric secretions, tears, urine, bile, milk, semen, amniotic fluid and some pathological fluids as well known as secretors whereas others who lack this property are called non-secretors [3,4]. The antigens expressed on both the red cells and in the secretions are determined by the interaction of Hh, Sese, ABO and Lele genes[1,2]. The present study was carried out to find out the association of Lewis blood groups in peptic ulcer disease patients with secretor status.

Materials and methods

Fifty endoscopically diagnosed peptic ulcer patients were selected from medical units of Services Hospital, Sir Ganga Ram Hospital, and Mayo Hospital. Another 50 individuals from blood bank were taken as a control. Lewis blood grouping was done on blood samples of patients and controls by using anti-Le^a and anti-Le^b anti sera by direct agglutination test[5]. One ml non-stimulated saliva was used for secretor status analysis by Wiener agglutination inhibition test adopted by Vidas et al[4]. Statistical analysis of Chi square test and "p" value were used to analyze the results and data in the present study.

Results

The results are given in tables 1,2 3 & 4.

Table 1.Lewis Phenotype in Controls and PUD

Lewis	PUD %	Controls %
Le (a+b-)	22	16
Le (a-b+)	72	66
Le (a-b-)	04	16
Le (a+b+)	02	02
Total	100	100

(χ^2 =2.9858 with 3df, p >0.1).

Table 2. Frequency of Lewis blood groups in male and female controls and PUD patients

Lewis phenotype	Male		Female	
	Control No(%)	PUD No (%)	Control No(%)	PUD No (%)
(a+b-)	5(14.7)	7(20.6)	3(18.8)	4(2.5)
(a-b+)	22(64.7)	25(73.6)	11(68.7)	11(68.7)
(a-b-)	6(17.7)	1(2.9)	2(12.516)	1(6.3)
(a+b+)	1(2.9)	1(2.9)	0	0
Total	34(100)	34(100)	16(100)	16(100)

Table 3. Frequency of male and female secretor status in PUD patients and controls

Secretor Status	PUD		Control	
	Male No(%)	Female No (%)	Male No(%)	Female No (%)
Secretor	27(79.4)	11(68.7)	28(82.4)	12(75)
Non secretor	7(20.6)	5(31.25)	6(17.6)	4(25)
Total	34(100)	16(100)	34(100)	16(100)

Association of male and female sex with secretor status
χ^2=0.5829, df=1, p>0.5 (Non-significant)

Table 4. Secretor Status.

Secretors status	PUD%	Controls%
Secretor	76	80
Non-secretor	24	20

Discussion

Lewis blood grouping was never done in this country and therefore no data is available for Pakistani population. The results between controls and PUD patients are compared in table 1. PUD patients showed predominance of blood group Le (a-b+) but this Lewis phenotype is not associated with duodenal ulcer (χ^2 =2.9858 with 3df, p >0.1).

No broad based statistical data is available on secretor status of Pakistani population. In a study Rizvi et al[3] found that secretors and non-secretors are 85.3% and 14.7% respectively. The male secretors were 81.6% as compared to 90.9% of the female secretors. In another study Rizvi et al[6] found 56% secretor and 44% non-secretor in duodenal ulcer patients. Hook-Nikanne et al[7] found 80% of blood donors as secretor and 20% as non-secretors in Helsinki urban population. Lamey et al[8] (1994) found secretor as 64% and non-secretors as 36% in Sri-Lankan population.

The ability of secreting the blood group antigens plays a significant role in the natural resistance of the organism to infection. Bacterial colonization and ensuing inflammatory response may be influenced by the host expression of Lewis blood group antigens[9]. Helicobacter pylori colonization and ensuing inflammatory response is more in Lewis-b patients[10].

Lewis antigens are considered protective as well as receptors for infection. The H type 2, Lewis b (Le^b) and Lewis a (Le^a) antigens can act as receptors for H. pylori on the gastric mucosa but H type 2 (the antigen of blood group O) appears to be more efficient receptor for the 61-kDa adhesion than are the Lewis antigens[10]. The Lewis b antigen in the Lewis blood group mediates H. pylori attachment to gastric mucosa[11]. Alkout et al[10] suggested that H type 2, found on almost all individuals is a key receptor of H pylori. Lewis b binds H pylori more efficiently than Lewis a. Both secretor and non-secretor express H type 2 but non-secretors lack Lewis b.

Other workers failed to define any association between infection H pylori and blood group or secretor status[7,12,13]. Predisposition to H. pylori gastric antral infection is not associated with Lewis blood group and secretor status[13]. All these studies confirm the results of present study results that Lewis phenotypes are not associated with peptic ulcer disease or duodenal ulcer.

Further carefully controlled studies are needed to find relationship between H. pylori, peptic ulcer disease and Lewis blood group antigens. This area has future potential of interest and advancement.

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