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Study of Blood Group (ABO and RH) Association and Secretor Status of ABH Substances in Patients with Systemic Lupus Erythematosus

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Abstract: *Individuals who secrete their blood group antigens in the body fluids are called secretors and individuals who do not secrete are called non-secretors. Non-secretors of blood group antigens have been found to be associated with many infectious and autoimmune diseases by many previous studies. The aims of our study were to find out any association between ABO blood group, RhD blood group and secretor status of ABH substances with the patients of Systemic Lupus Erythematosus. This Case Control study was carried out in the Department of Transfusion Medicine in collaboration with the Department of Medicine, Regional Institute of Medical Sciences, Imphal Manipur from September 2016 to August 2018. 75 confirmed SLE patients attending Rheumatology OPD, Department of Medicine and 75 healthy sex matched voluntary blood donors attending blood bank, Department of Tranfusion Medicine have been enrolled in the study. Their blood were typed for ABO and RhD groups using conventional tube technique and saliva were tested for secretor and non-secretor status using heamagglutination inhibition test. In SLE group, there were 45.3% with A blood group followed by O (36%), B (14.7%) and AB (4%) and in non-SLE group 36% were with A blood group followed by O (29.3%), B (22.7%) and AB (12%). There were no significant association between SLE status and ABO blood groups (p= 0.133). Majority of participant in SLE were A RhD +ve (45.3%) followed by O RhD +ve (34.7%), B RhD +ve (14.7%), AB RhD +ve (4%) and O RhD –ve (1.3%) and in non-SLE group 36% were A RhD +ve followed by O RhD +ve (28%), B RhD +ve (22.7%), AB RhD +ve (9.3%) and O RhD –ve (1.3%). A non-significant p value (p=0.285) infers that there is no significant association between RhD blood group and SLE.Among individual with SLE, 40% were non-secretors and in non-SLE only 14.7% were non-secretors. Therefore the odds of non-secretors develop SLE is 3.86 times when compared to non-SLE group. Therefore, it appears that non-secretor individuals are more prone to develop SLE.*

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I. Introduction

Systemic lupus erythematosus (SLE) is a chronic, relapsing, often febrile multisystemic autoimmune disorder affecting principally the skin, joints, kidneys and serosal membranes and lacking a single unifying pathognomic marker. The prevalence rate in India is approximately 1 in 2000 of the population. The term lupus means wolf in Latin and originally denoted wolf bite like facial lesions. Cazenave in France coined the term 'lupus erythematosus' in 1833.1 Systemic lupus erythematosus is an autoimmune disease in which organs and cells undergo damage initially mediated by tissue-binding autoantibodies and immune complexes. In most patients, autoantibodies are present for a few years before the first clinical symtoms appears; clinical manifestation are heterogeneous. Ninety percent of patients at diagnosis are women of childbearing years; people of all genders, ages, and ethnic groups are susceptible. The diagnosis of SLE is based on characteristic clinical features and autoantibodies. Any combination of $\geq$4 of 11 diagnostic criteria well documented at any time during an individual's history makes it likely that the patient has SLE (specificity and sensitivity are 95% and 75% respectively). In many patients criteria accrue over time. Antinuclear antibodies (ANA) are positive in >98% of patients during the course of disease, repeated negative tests suggest that the diagnosis is not SLE unless other autoantibodies are present. High titres IgG antibodies to double-stranded DNA and antibodies to the Sm antigen are both specific for SLE and therefore favour the diagnosis in the presence of compatible clinical manifestation.2 Individuals can be classified as secretors and non-secretors according to their ability to secrete

ABH blood group antigens in body fluids such as saliva, tears, urine, breast milk and bile etc. ABH secretions are controlled by fucosyltransferase 2 (FUT2) secretor gene located on the short arm of chromosome number 19 in the form of two allelic genes denoted as dominant 'Se' and recessive 'se'. 'Se' gene product is an L-fucosyltransferase that preferentially adds L-fucose to the type-1 oligosaccharide chain structures and responsible for the expression of soluble H substance in the body secretion such as saliva. An individual who inherits the 'Se' allele in either homozygous (SeSe) or heterozygous (Sese) manner is classified as secretor and an individual who inherit the 'se' allele in homozygous (sese) manner is classified as non-secretor. Therefore, O secretor will secrete H substance, A secretor will secrete A and H substances while B secretor will secrete B and H substances in the body fluids.The 'se' allele is an amorph and homozygous expression does not convert glycoprotein precursor to soluble H substance and has neither soluble H antigens nor soluble A or B antigens present in the body fluids.About 80% of the random population inherit the 'Se' allele and is classified as secretor and about 20% of the random population can be considered non-secretors.3 The present study was taken up to study the association of blood group (ABO and Rh) and ABH secretor status with the patient of SLE. The knowledge will help Clinicians and Rheumatologist for better understanding and diagnosis of SLE in this region of the country.

II. Material And Methods

The aims of our study were to find out any association between ABO blood group, RhD blood group and secretor status of ABH substances with the patients of Systemic Lupus Erythematosus. This Case Control study was carried out in the Department of Transfusion Medicine in collaboration with the Department of Medicine, Regional Institute of Medical Sciences, Imphal Manipur from September 2016 to August 2018. 75 confirmed SLE patients attending Rheumatology OPD, Department of Medicine and 75 healthy sex matched voluntary blood donors attending blood bank, Department of Tranfusion Medicine have been enrolled in the study.

Inclusion criteria: The patients who have been diagnosed as having SLE as confirmed by clinical history and laboratory tests were included in the study. All the cases whether treatment has started or not, were taken up for the study.

Exclusion criteria:1.Patients having SLE like symptoms but not fulfilling the diagnostic criteria for SLE.2.Patients who are unable to collect saliva and not willing to participate.3.Drug induced lupus.

Study Tools:1.Brief clinical history and laboratory diagnosis were taken.2.Laboratory investigations.The under mentioned laboratory investigation were performed in the Department of Transfusion Medicine, RIMS.1.ABO and Rhesus grouping by using standard cell grouping tube method.2.Haemagglutination inhibition test for detection of ABH substances in saliva of the patient.3.A proforma design for the study was prepared and all necessary data for analysis were entered.

Statistical analysis: Data were entered in Microsoft Excel and analysed using Stata version 14. Age was expressed as mean (standard deviation). The difference in mean age between the SLE and non-SLE groups was analysed using independent t test. The distribution of gender and age categories expressed as percentages and its difference in distribution between the groups were assessed using chi-squared test. The association of ABO blood group, RhD blood group and secretor status between the SLE and non-SLE group were assessed using chi-square test. A p value of < 0.05 was considered as statistically significant.

Ethical issue: Ethical approval was obtained from the Research Ethics Board, RIMS, Imphal, before starting the study.Consent from the participant were obtained and confidentially were maintained.

III. Result

Distribution of age between SLE and non-SLE group infers that there is no significant difference between the two groups (p-value = 0.166) (Table-1). But the mean (SD) age of the participants in SLE group was 34.15 (11.4) and in non-SLE group mean (SD) age was 23.57 (6.69) years. There was a significant difference in mean age between the two groups (Table-2). There were 74 (98.7%) female participants and 1 (1.3%) male in SLE group. All participants in non-SLE groups were females. The group were comparable based on gender (p value=1) (Table-3). In SLE group, there were 34 (45.3%) participants with blood group A, 27 (36%) with O group, 11 (14.7%) with B group and 3 (4%) with AB blood group. In non-SLE group 27 (36%) were with blood group A, 22 (29.3%) with O group, 17 (22.7%) with B group and 9 (12%) were with AB blood group. There were no significant association between SLE status and ABO blood groups (p value=0.133)

(Table-4). Association of RhD blood group with SLE represents majority of the people were A positive (45.3%) followed by O positive (34.7%), B positive (14.7%), AB positive (4%) and O negative (1.3%) respectively in SLE group when compared to non-SLE group where there was a distribution of A positive (36%), O positive (28%), B positive (22.7%), AB positive (9.3%) and O negative (1.3%). A non-significant p value (p=0.285) infers that there is no significant difference in Rh blood groups between SLE and non-SLE individuals (Table-5).Association of ABH secretor status with SLE represent that 60% in SLE group are secretors of ABH blood group substance while 85.3% in non-SLE are secretors. Among individual with SLE 40% were non-secretors while in non-SLE only 14.7% were non-secretors and the odds of non-secretor develop SLE is 3.86 (95% CI 1.762, 8.538) times compared to non-SLE group. There is a significant association between the secretor status and SLE with a p-value of less than 0.001 (Table-6).

Table-1: Distribution of age between the SLE and Non-SLE groups.

SLE Status	Age category		P Value
	>45 years N (%)	<45 years N (%)	
SLE	7 (9.3%)	68 (90.7%)	0.166
NON-SLE	2 (2.7%)	73 (97.3%	
Total	9 (6%)	141 (94%)	

Table-2: Distribution of mean (SD) age between the SLE and Non SLE groups.

SLE Status	Number	Mean Age	Std. Deviation	t value	P value
SLE	75	34.15	11.416	6.9	<0.001
NON-SLE	75	23.57	6.698		

Table-3: Distribution of gender between the SLE and Non-SLE groups.

SLE Status	Gender		P value
	Female N (%)	Male N (%)	
SLE	74 (98.7%)	1 (1.3%)	1
NON-SLE	75 (100%)	0	
Total	149 (99.35)	1 (0.7%)	

Table-4: Association of ABO blood groups with SLE status.

		ABO GROUP				Chi-Square value	P value
		A	B	O	AB		
SLE STATUS	SLE	34 (45.3%)	11 (14.7%)	27 (36%)	3 (4%)	5.6	0.133
	NON-SLE	27 (36%)	17 (22.7%)	22 (29.3%)	9 (12%)		
	Total	61 (40.7%)	28 (18.7%)	49 (32.7%)	12 (8%)		

Table-5: Association of RhD blood groups with SLE status.

	RhD GROUP						X^2 Value	P value
	A+ve	B+ve	O+ve	AB+ve	O -ve	AB-ve		
SLE	34 (45.3%)	11 (14.7%)	26(34.7%)	3 (4%)	1 (1.3%)	0	0.621	0.285
NON-SLE	27 (36%)	17 (22.7%)	21 (28%)	7 (9.3%)	1 (1.3%)	2 (2.7%)		
Total	61 (40.7%)	28 (18.7%)	47(31.3%)	10 (6.7%)	2 (2.7%)	2 (2.7%)		

Table-6: Association of secretor status with SLE status.

		SECRETOR STATUS		Chi-Square value	P value
		Non secretor	Secretor		
SLE STATUS	SLE	30 (40%)	45 (60%)	12.1	<0.001
	Non SLE	11 (14.7%)	64 (85.3%)		
	Total	41 (27.3%)	109 2.7%)		

IV. Discussion

In our study in the SLE group, there were 45.3% with blood group A, 36% with blood group O, 14.7% with blood group B and 4 % with blood group AB and in non-SLE group, there were 36% with blood group A, 29.3% with O, 22.7% with B and 12% with AB. There were no significant association between SLE and ABO blood group status (p value=0.133). Similarly, Tamega AA et al[4] studied the association between blood groups (ABO and Rh) and Discoid Lupus Erythematosus. There was no significant difference between the frequency of blood groups from Discoid Lupus patients and local population. In another similar study conducted by Naorem S et al[5] to determine the distribution of ABO and Rh blood group in patients with Rheumatoid Arthritis and to

correlate the severity of Rheumatoid Arthritis with blood group phenotypes. Their study shows no significant correlation between ABO blood group and Rheumatoid Arthritis. In the study of Ozyurt K et al[6],the distribution of ABO and Rhesus blood groups in patients with Behcet's disease was similar to the healthy donors and no relationship was interpreted. Similarly Sambo N et al[7] studied a total 225 individuals of age 17-90 years consisting of 100 confirmed diabetic patients and 125 non-diabetic individuals. The findings in their study suggested that there was no association between ABO blood group and Diabetes mellitus. However, study by Bakhtiari S et al[8] studied the relationship between secretion or non-secretion of blood group antigens into the saliva and oral lichen planus and it was found that a large majority of the people examined in both groups were secretors of blood group A, however most OLP patients belonged to blood group B.Similarly, Odaibo GN et al[9] studied association between HIV status and ABO blood group but the findings suggested no association between ABO blood groups and HIV infection. Likewise, Igbeneghu C et al[10] studied 158 malaria and 182 control subjects in order to determine the association between ABO blood group, secretor status and Malaria infection. Malaria among that study population was not associated with ABO blood group. However in a study by Mattos LC et al[11] where the frequencies of ABO, Lewis blood group phenotypes, secretor and non-secretor phenotype in patients infected or uninfected by H. Pylori were evaluated, results showed that blood group A was (27.0%), B (12.2%), AB (4.0%) and O (56.8%) among infected and blood group A was (58.7%), B (13.0%), AB (4.3%) and O (24.0%) among uninfected patients. The difference showed was significant. Our study interests to determine the association of Rh(D) blood groups with SLE results represents majority of the people were in A positive (45.3%) followed by O positive (34.7 %), B positive (14.7%), AB positive (4%) and O negative (1.3%) in SLE group when compared to non-SLE group where there was a distribution of A positive (36%), O positive (28%)), B positive (22.7%), AB positive (9.3%) and O negative (1.3%). There was no significant difference in RHD blood groups between SLE and non-SLE individuals (p=0.285). Similar study by Tamega AA et al[4] that determined the association between blood groups (ABO and Rh) and Discoid Lupus Erythematosus.Their study exhibited no significant correlation between ABO-Rh blood groups and Discoid Lupus Erythematosus. Similarly, Ozyurt K et al[6] studied the distribution of ABO and RhD blood groups in patients with Behcet's disease and healthy donors and no significant association was found between the cases and control group in the study conducted. In our study the percentage of non-secretors was 40% in SLE patients and 14.7% in non-SLE. The percentage of secretors and non-secretors varies with the SLE and non-SLE population. Non-secretor status in patients with SLE was more frequent compared with non-SLE, 30 out of 75 SLE patients Vs 11 out of 75 non-SLE ($p< 0.001$). A similar kind of results was observed in a study conducted by Shahidi-Dadras M et al[12] among patients with Oral Lichen Planus, where non-secretor individuals were more prone to OLP. Similarly another study by Shahidi-Dadras M et al[13] studied 50 Psoriasis patients and 100 age and gender match control subject. Their study concluded that non-secretors are at risk of developing Psoriasis. Likewise Shahidi-Dadras M et al[14] studied the frequency of secretor status and Lewis phenotypes in patients with Pemphigus vulgaris compared with healthy controls in order to determine their roles in this autoimmune disease. Based on their study they have concluded that non secretors individuals were more susceptible to Pemphigus vulagaris. Like our study, A study conducted by Sambo N et al[7] among diabetic and non-diabetic individuals also found a similar kind of observation where non-secretors were significantly more associated with diabetes than secretors (X^2=6.953,df=1,p=0.005). The study by Dayaprasad G et al[15] to determine association of secretor status with vaginal candidiasis and study by Olorunshola KV et al[16] with sickle cell disease showed a similar pattern with higher prevalence of disease among non-secretors. Doll R et al[17] reported a similar finding to our study findings in duodenal ulcer, gastric ulcer, and stomach ulcer patients. For duodenal ulcer the results show that the relative incidence among non-secretors compared with secretors is 1.80 to 1, with 95% confidence limits of 1.55 to 1 and 2.03 to 1. For gastric ulcer the relative incidence is 1.42 to 1, with 95% confidence limits of 1.16 to 1 and 1.74 to 1. Comparison of the results for the two types of peptic ulcer shows that they are significantly different from one another. Similarly in our study we found that the secretors and non-secretors are significantly different in SLE and non-SLE population.However, findings of an investigation conducted by Odaibo GN et al[9] showed that the Secretors were significantly more associated with HIV infection than non-secretors (χ^2=7.953, df=1,p=0.005). ABO blood group was not significantly associated with HIV infection (χ^2 =1.66, df =2, p=0.558). There was a significant association between group O and secretor in controls (χ^2 = 5.964, df=1, p= 0.015) but not in HIV infection (χ^2 =0.004, df=1, p=0.949). These findings suggest that while there was no association between ABO blood groups and HIV infection, secretion of ABH antigens is associated with HIV infection.In contrast to our study, a study conducted by Jaff MS et al[18] among 762 individuals reported that 76.1% of the study population was ABH secretor and 23.9% non-secretor. The difference in observation might be due to the variation in study population or due to the difference in technique used by the investigators to find out the secretor status. Another study by Bakhtiari S et al[8], assessed the relationship between secretion or non-secretion of blood group antigens into the saliva and Oral Lichen Planus. In the case group, 25 subjects (84.4%) were secretors and 5 (16.6%) were non secretors. In the control group, 24 subjects (80.0%) were secretors and 6 (20.0%) were non-secretors. There was no significant difference between the case and control groups for

secretor status (p= 0.73). They concluded that their study did not indicate a significant difference in salivary secretor status between OLP patients compared to controls. This difference in findings compared to our study may be due to the lack of adequate power for their study because of the smaller sample size.

V. Conclusion

In SLE group, there were 45.3% with A blood group followed by O (36%), B (14.7%) and AB (4%) and in non SLE group 36% were with A blood group followed by O (29.3%), B (22.7%) and AB (12%). There were no significant association between SLE status and ABO blood groups (p value=0.133). Majority of participants in SLE were A positive (45.3%) followed by O positive (34.7%), B positive (14.7%), AB positive (4%) and O negative (1.3%) and in non-SLE group 36% were A positive followed by O positive (28%), B positive (22.7%), AB positive (9.3%) and O negative (1.3%). A non-significant p value (p=0.285) infers that there is no significant association between RhD blood group and SLE. Among individuals with SLE, 40% were non-secretors and in non-SLE only 14.7 % were non-secretors. Therefore, the odds of non-secretors develop SLE is 3.86 (95% CI 1.762, 8.538) times when compared to non-SLE group. Therefore, it appears that non-secretor individuals are more prone to develop SLE.

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