ORIGINAL RESEARCH ARTICLE

LH-type Distribution in Diabetes Mellitus Patients and Nondiabetics

Avinash Tiwari[1], Anterpreet Arora[2], Shyamal Koley[3]

Abstract

Introduction: Diabetes mellitus is a major health disorder which is proving to be epidemic globally with alarming rate. The current study was done to find the of LH-type distribution in the patients of diabetes mellitus type-2.

Methods: The samples of blood of the diagnosed cases of diabetes mellitus type-2 ($n = 100$) were randomly collected from the laboratory of Department of Physiotherapy, Guru Nanak Dev University, Amritsar, India. They were typed for the both ABO and LH blood groups and compared with an equal number ($n = 100$) of appropriate controls.

Results: It was specified that patients suffering with diabetes mellitus of type-2 in comparison with the normal population considered as controls were overwhelmingly LH-negative with *p*-value highly significant ($p < 0.001$) and that this was chiefly noticeable in diabetes mellitus patients with A and B blood groups.

Conclusion: The persistence of hyperglycemic environment of the erythrocyte membrane in diabetes mellitus patients and saturation of the membrane receptors with glucose molecules leave less availability of receptors to get bound with the anti-LH lectin *Erythrina lithosperma*. So we have come to the conclusion from the results of the current study that the patients suffering from diabetes have blood group LH-negative in more individuals in comparison with the nondiabetics.

Keywords: ABO typing, Diabetes mellitus, LH specificity, LH typing.

AMEI's Current Trends in Diagnosis & Treatment (2021): 10.5005/jp-journals-10055-0136

Introduction

Strivastava et al.[1] revealed new red blood specificity in the seeds of a plant *Erythrina lithosperma* which belongs to Leguminosae family. The name LH was derived from Ludwik Hirszfeld, who was a serologist from Polland. More studies concerning the immunochemical properties, LH-specificity distribution, and genetics of the *E. lithosperma* lectin have been studied by numerous scientists.[2–12]

The anti-LH lectin reacts with human red cells by either clumping them firmly or just weakly agglutinating them. The former type of reaction is called LH-positive and the latter, LH-negative. Since the anti-LH lectin is inhibited by some sugars,[1] the difference between LH-positive and LH-negative types of reaction patterns may depend on the distribution of cellular lectin binding receptors, which are either similar or alike in structure to those carbohydrates. Therefore, it was believed that in several glycosylated RBCs from individuals of diabetes, there are scarcer receptors offered to lectin molecules on the surface of the cells to get bound, and as a result, there must the patients suffering from diabetes have blood group LH-negative in more individuals in comparison with the nondiabetics.

The lectin studies have immense importance in diabetes mellitus research. Still the studies of LH specificity in patients with diabetes mellitus are scanty, especially from the state of Punjab. Thus, the current study was intended to accomplish the knowledge and fill the voids in this field.

Materials and Methods

Samples

Informed consent was obtained from all the individuals participating in the current research. The samples of blood of the diagnosed cases of diabetes mellitus type-2 ($n = 100$) were randomly collected from the laboratory of Department of Physiotherapy, Guru Nanak Dev University, Amritsar, India. The patients chosen for taking samples were males and females of 40 years or more. The controls were normal healthy individuals whose samples were taken in a random manner. As the differences in sex do not affect the LH types, the samples from both the sexes were collected and assembled for investigation. The study was presented to the Institutional Ethical Committee and was approved by it.

ABO Typing

The fingertip was cleaned and sterilized then given a prick with lancet fully sterile. Blood is collected in test tube with 0.85% NaCl. RBC pellet was washed three times with 0.85% NaCl by getting centrifuged at 2500–3000 rpm. The suspension of 2% RBCs was prepared in 0.85% NaCl. Later by using standard serological method with 2% RBCs suspension and anti-A and anti-B antisera, ABO typing was done.

[1,3]Department of Physiotherapy, Guru Nanak Dev University, Amritsar, Punjab, India

[2]Department of Anatomy, SGRDIMSAR, Amritsar, Punjab, India

Corresponding Author: Anterpreet Arora, Department of Anatomy, SGRDIMSAR, Amritsar, Punjab, India, Phone: +91 9814975545, e-mail: anterpreetarora1@gmail.com

How to cite this article: Tiwari A, Arora A, Koley S. LH-type Distribution in Diabetes Mellitus Patients and Nondiabetics. AMEI's Curr Trends Diagn Treat 2021;5(2):89–91.

Source of support: Nil

Conflict of interest: None

Anti-LH Lectin

With the method described by Shrivastava et al.,[1] we did LH typing with the anti-LH lectin attained from the *E. lithosperma* seeds in Physiotherapy laboratory of Guru Nanak Dev University. The *E. lithosperma* seeds were acquired from Kolkata from Botanical Survey of India.

Anti-LH Lectin Preparation

In normal saline from seed extracts of *E. lithosperma,* anti-LH lectin was formed. The extracts were grinded to powder in a fine manner and mixed with saline solution, the ratio being 1:9. The mixture was kept at normal temperature for 4 hours, with intermittent stirring. After this period, centrifugation of the slurry was done at 3000–4000 rpm for 30 minutes. Afterward, the supernatant which turned clear was separated and kept in refrigerator. The addition of sodium azide was done to it in the ratio of 1:10000 for preservation.

LH Typing

One drop of anti-LH lectin (*E. lithosperma*) and four drops of 2% suspension of RBCs from a Pasteur pipet was added in normal saline. The normal saline was contained in a serological test tube of 15 mm × 15 mm measurement. The blend was the centrifuged for 20 seconds at 2500 rpm. Formation of a cell button occurred in all cases, but in some abrupt shaking of the tube, it resulted in dispersion of the cell button into a many agglutinates of small size. This reaction was called LH-negative, whereas if the tube was shaken similarly and the cell button kept undamaged, the reaction was considered as LH-positive. The checking of the outcomes of the tube test is done by the titration method. The range of titer LH-positive cells was 32–64, and in case of LH-negative cells, it was 8–16.[13]

Analysis of the Data—Statistics

The distribution of frequency of ABO and LH typing was followed by Chi-square test for the correlation of LH typing with ABO blood type. Software Statistical Package for Social Science (SPSS) of version 20.0 was used for the analysis of the data. To show statistical significance, 5% of probability was used.

Results

The phenotype distribution of the LH types and allele frequencies in diabetes mellitus patients and normal individuals (controls) is shown in Table 1. The allele frequencies are higher in diabetes mellitus patients (43.00%) in LH-negative type than normal individuals (31.00%). The differences between the patients and controls in regard to the distribution of LH-negative type are highly significant (p <0.001). In patients, the allele frequency of LH-negative type is 0.2433, whereas in controls, it is 0.1693. Both types of population are considered in Hardy-Weinberg equilibrium.

Table 2 displays the distribution of ABO blood types along with their allele frequencies in patients with diabetes mellitus and controls. Patients have a little higher frequency in 26.00, 7.00, 27.00 and 40.00%, respectively. No significant variation (p >0.05) exists between them. The patients have higher allele frequencies in A (0.2208) and B (0.2869) than controls. Both the populations are considered in Hardy-Weinberg equilibrium.

The LH-type distribution in A blood group in diabetes mellitus patients and controls is given in Table 3. Diabetes mellitus patients have higher frequency (69.23%) in LH-negative type than controls (44.44%). The differences between the diabetes mellitus patients and controls in regard to the distribution of LH-negative type with blood group A are highly significant (p <0.001). Both types of population are considered in Hardy-Weinberg equilibrium.

Table 4 shows LH-type distribution and allele frequencies in diabetes mellitus patients and controls with B blood group. Patients have higher frequency (51.28%) in LH-negative type than controls (35.00%). The differences between the patients and controls in regard to the distribution of LH-negative type with blood group-B are highly significant (p <0.001), although both types of population are considered in Hardy-Weinberg equilibrium.

Table 5 shows LH-type distribution and allele frequencies in diabetes mellitus patients and controls with AB blood group. Patients have higher frequency (66.66%) in LH-negative type than controls (42.86%). The differences between the patients and controls in regard to the distribution of LH-negative type with blood group A are not significant (p >0.05), although both types of population are in Hardy-Weinberg equilibrium.

The LH types are invariably LH-positive in blood group O in all cases both in patients and controls.

Discussion

This aim of this study was to discover and know the LH specificity differs in patients with type-2 diabetes mellitus and in normal individuals

Table 1: LH-type distribution and allele frequencies in diabetes mellitus patients and controls

			Phenotype frequencies		*Allele frequencies*		*Hardy-Weinberg equilibrium*
Sample	*n*		LH^+	LH^-	LH^+	LH^-	
Patients	100	Obs. Nos	57	43	0.7567	0.2433	0.0000
		Obs.%	57.00	43.00			
Controls	100	Obs. Nos	69	31	0.8307	0.1693	0.0000
		Obs.%	69.00	31.00			

Chi-square (df.1) = 3.086; p <0.001

Table 2: ABO-type distribution and allele frequencies in diabetes mellitus patients and controls

			Phenotype frequencies				*Allele frequencies*			*Hardy-Weinberg equilibrium*
Sample	*n*		*O*	*A*	*B*	*AB*	*A*	*B*	*O*	
Patients	100	Obs. Nos	25	27	39	9	0.2208	0.2869	0.4922	3.6706
		Obs.%	25.00	27.00	39.00	9.00				
Controls	100	Obs. Nos	27	26	40	7	0.2094	0.2784	0.5120	1.7382
		Obs.%	27.00	26.00	40.00	7.00				

Chi-square (df. 3) = 0.356; p >0.05

Table 3: LH-type distribution and allele frequencies in diabetes mellitus patients and controls with A blood group

Sample		*n*	*LH*$^+$	*LH*$^-$	*Hardy-Weinberg equilibrium*
Patients	26	Obs. Nos	8	18	0.0000
		Obs.%	30.76	69.23	
Controls	27	Obs. Nos	15	12	0.0000
		Obs.%	55.55	44.44	

Chi-square (df. 1) = 3.307; p <0.001

Table 4: LH-type distribution and allele frequencies in diabetes mellitus patients and controls with B blood group

Sample		*n*	*LH*$^+$	*LH*$^-$	*Hardy-Weinberg equilibrium*
Patients	39	Obs. Nos	19	20	0.0000
		Obs.%	48.72	51.28	
Controls	40	Obs. Nos	26	14	0.0000
		Obs.%	65.00	35.00	

Chi-square (df. 1) = 2,136; p <0.01

Table 5: LH-type distribution and allele frequencies in diabetes mellitus patients and controls with AB blood group

Sample		*n*	*LH*$^+$	*LH*$^-$	*Hardy-Weinberg equilibrium*
Patients	9	Obs. Nos	3	6	0.0000
		Obs.%	33.33	66.66	
Controls	7	Obs. Nos	4	3	0.0000
		Obs.%	57.14	42.86	

Chi-square (df. 1) = 0.912; p >0.05

without diabetes. This concept was generated on the conjecture that consistency of hyperglycemia in patients with diabetes mellitus either because of inadequate secretion of insulin or inadequate consumption of the glucose, there exists hyperglycemic environment for erythrocytes for a long time. Consequently, there are substantial changes in aggregation and deformity of erythrocytes. The reports also indicate that the in diabetes mellitus patients, spectrin is oxidatively impaired and there is extreme glycosylation of cytoskeleton proteins of the erythrocytes.[14] Moreover, we supposed that excessive *in vivo* glycosylation of erythrocyte membrane should give a fewer number attachment sites to the carbohydrate-rich reactive component of the anti-LH lectin as compared to the other erythrocytes. Thus, in diabetes mellitus patients, LH-negative individuals are more as compared to the nondiabetics. The outcomes of the current research direct that the patients with diabetes mellitus are exceedingly LH-negative than the normal individuals (p <0.001) and also that this variation is mainly conspicuous in diabetes mellitus with A and B blood groups. A substantial dominance of LH-negative type in diabetes mellitus patients with A and B blood groups directs us to a conclusion that the LH system has more than a chance association with diabetes mellitus. The O individuals were not considered because they were all LH-positive. The reaction of AB cells is weak with the anti-LH lectin in the order O > B > A > AB, and this enlightens why significant variation in the distribution of the LH types in diabetes mellitus patients and controls with blood group AB was not established. The results of the current research followed the findings of previous studies.[1,6,10] The striking results of the current study were that individuals with O blood group were customarily LH-positive.

Conclusion

The persistence of hyperglycemic environment of the erythrocyte membrane in diabetes mellitus patients and saturation of the membrane receptors with glucose molecules leave less availability of receptors to get bound with the anti-LH lectin *E. lithosperma*. So we have come to the conclusion from the results of the current study that the patients suffering from diabetes have blood group LH-negative in more individuals in comparison with the non-diabetes mellitus patients.

References

1. Shrivastava PK, Sehajpal PK, Kaur H. The LH system: a new red blood cell membrane specificity in man. Acta Anthropogenet 1979;3:83–92. NLM ID: 7801158.
2. Shrivastava PK, Sehajpal PK. Genetics of the LH red blood cell membrane specificity. Acta Anthropogenet 1980;4(1–2):85–88. PMID: 7306377.
3. Shrivastava PK, Sehajpal PK. Distribution of LH types in some north Indian populations. Acta Anthropogenet 1981;5(3):181–185. PMID: 7272004.
4. Reddy VR, Naidu GP, Shrivastava PK. The LH red blood cell membrane specificity in three south Indian populations. Acta Anthropogenet 1981;5(3):189–194. PMID: 7272005.
5. Kaur K. The LH red blood cell membrane specificity in man and selected animals [PhD thesis]. Patiala: Punjabi University; 1983.
6. Koley S, Jain R, Shrivastava PK. The LH-specificity in diabetes mellitus. In: Bansal IJS, et al., editors. New horizons in human biology. New Delhi: Today and Tomorrow's Printer and Publishers; 1991.
7. Koley S, Jain R, Shrivastava PK. The distribution of the ABO and LH blood groups in some central Indian populations. J Hum Ecol 1992;3(3):239–242. DOI: 10.1080/09709274.1992.11907931.
8. Koley S. The distribution of the LH types among the Kshatriya patients with diabetes mellitus in Sagar (M.P.). Madhya Bharty J 1994;34A–38B Part II:7–12.
9. Koley S. The LH-specificity in the Brahmin patients with diabetes mellitus in Sagar (M.P.). J Ravishankar Univ 1996;9(1):25–30.
10. Koley S, Jain R, Shrivastava PK. Serum antibody titres against LH-positive and LH-negative red blood cells. South Asian Anthropol 1992;13:43–45.
11. Koley S, Shrivastava PK. Four important properties of the anti-LH lectin. South Asian Anthrop 1993;14:51–54.
12. Koley S, Shrivastava PK. The distribution of ABO and LH blood types among four caste groups in patients with diabetes mellitus. South Asian Anthropol 1993;15:41–46.
13. Koley S. Segmental differences in males and females: analysis of secondary data. Indian Sports Studies 2003;2(1):25–29.
14. Shin S, Ku Y, Babu N, et al. Erythrocyte deformability and its variation in diabetes mellitus. Indian J Exp Biol 2007;45(1):121–128. PMID: 17249336.