

Frequency of ABO Blood Groups in HIV Infected Individuals in Khammam-Telangana

A. Purna Singh¹, Bagavathiammal Periyasamy², Rajput S. A. Kiran Singh³, Seralathan G⁴, Thamizhvanan P⁵, Nallasivam P⁵, Panneerselvam Periasamy⁶

¹Associate Professor, Department of Physiology, Mamata Medical College, Khammam, Telangana, India

²Associate professor, Department of Biochemistry, Swamy Vivekanandha Medical College Hospital and Research Institute, Tiruchengode Namakkal, Tamil Nadu, India

³Associate professor, Department of Pathology, Dr Patnam Mahender Reddy Institute of Medical Sciences, Hyderabad, Telangana, India

⁴Assistant Professor, Department of Physiology, Government Erode Medical College, Perundurai, Tamilnadu, India

⁵Tutor, Department of Physiology, Government Erode Medical College, Perundurai, Tamilnadu, India

⁶Assistant Professor, Department of Physiology, Government Erode Medical College, Perundurai, Tamilnadu, India

Received: 25-12-2023 / Revised: 23-01-2024 / Accepted: 26-02-2024

Corresponding Author: Dr. Panneerselvam Periasamy

Conflict of interest: Nil

Abstract:

Introduction: HIV is a dreadful disease in present society. However, everyone is not infected (even after Transfusion of Contaminated blood). There must be something protecting the infection in some of the individuals of given population. It is likely that the blood groups of an individual may be playing this role.

Aim: The present work is carried out to find whether HIV infection is correlated to blood groups of an individual.

Materials and Methods: The study was conducted between 2021-2023. Institutional Human Ethical approval has been taken. A total of 451 controls and 912 HIV infected individuals subjects were investigated for blood group status. Blood grouping is done by slide method.

Results: Cross tabulation and Analyses were done using the non-parametric chi-square test (p-value two sided) p value of < 0.05 was considered statistically significant.

Conclusion: When ABO blood group system is correlated with HIV infection, blood group 'B' individuals are more prone to develop HIV infection and blood group 'AB' individuals are least affected. HIV infection is not related to inheritance of Rhesus factor. These results infer that the severity of the disease in HIV infection is modified by the genetic constitution.

Keywords: Human Immunodeficiency Virus, Blood groups.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Human immunodeficiency virus (HIV) belongs to a member of Lentivirus and part of Retrovirus Family [1]. When a human being gets infected with HIV the immune system gradually fails resulting in susceptibility to various opportunistic infections and cancerous conditions [2]. The main source of infection is by contact with blood/blood products, Seminal fluid, vaginal fluid, unsafe sex, contaminated needles (especially in health care workers and drug addicts) and from mother to child through trans placental barrier and breast milk [3].

HIV infects vital cells of human immune system including the CD4 + T cells, macrophages, and dendrite cells. Most untreated people infected with HIV eventually develop AIDS [4]. These in-

dividuals usually suffer from severe morbidity attributed to opportunistic infections which are associated with the failure of immune system [5]. HIV progression to AIDS is influenced by viral, host, and environmental factors; where majority of the infected individuals will progress to AIDS within 8-10 years, few will progress much sooner (<5 years), and some will take much longer (> 15 years) to develop AIDS after HIV infection[6]. Treatment with anti-retroviral agents increases the life expectancy of people infected with HIV.

Even after HIV has progressed to diagnosable AIDS, the average survival time with antiretroviral therapy was estimated to be more than 5 years as of

2005 when compared to those not initiated on antiretroviral therapy typically die within a year [7][8]. The first case of HIV and AIDS was reported in 1981 [9] in Los Angeles and New York in a Homo sexual man. In 1983, researchers in the United States and France described the virus that causes AIDS [10], now known as HIV, belonging to the group of viruses called Retroviruses. While HIV infection results in the development of AIDS, the actual definition of AIDS is the reduction of T CD4 + cell counts below <200 cells/mm³.

The ABO Blood grouping system was discovered by the Austrian scientist Karl Landsteiner, who demonstrated three different blood types (A, B and O) in 1900 for which he was awarded Nobel Prize in 1930 [11][12]. Alfred Von Decastello and Adriano Sturli discovered the fourth blood group type 'AB' in 1902 [13]. Blood groups form a comparatively small field of study but have been recognized as having significant association with human genetics, immunology, anthropology, Clinical medicine and forensic medicine [14].

A strong Association has been described between Blood group O and Peptic ulcer [15], Blood group AB and Carcinoma cervix [16], Blood group A and Gastric Carcinoma [17], Blood group B and Pemphigus and Seborrhoric Dermatitis [18]. Up to now there is no study performed to determine the association of blood groups and susceptibility to HIV infection. So, we made an attempt to find the possibility of relationship between HIV infection and blood groups.

Materials and Methods

The study was conducted between 2008 and 2010. HIV infected individuals consent was taken. Institutional Human Ethical approval has been taken. Blood samples were collected from 912 HIV infected individuals of both the sexes with different age groups attending OPD of Dermatology of Govt. General Hospital and Mamata General Hospital, Khammam, Telangana, India, and volunteered for the present study. All HIV seropositive patients were screened and found reactive for antibodies against HIV by commercially available ELISA. The study included a control group 451 subjects who were patient's attendants at the Government Hospital, Khammam, Telangana, and who were not the relatives of the subjects and consisting of both the sexes of different age groups. All subjects were found to be physically fit and Non-reactive to HIV ELISA test.

Blood grouping: The Blood grouping was done by Open slide Technique. The Anti-A, Anti-B and Anti-D were obtained from Span diagnostics Ltd., Surat, India. The anti-serum was stored in refrigerator at 2-80 C when not in use. Open slide technique was used because of the simplicity in

handling the test and faster results. Agglutination is rapid on flat or concave surfaces, and hence less time consuming [19]. The test was carried out at room temperature, because it is the optimum for normal agglutinating type of anti-A or anti-B. In addition to descriptive analyses non-parametric Chi 2 – tests (p-value two sided) were used for comparisons.

Results

The Distribution of ABO blood groups as in controls in people of Khammam district is as follows (Table no.1); blood group 'O' is 181/451(40.13%), blood group 'A' is 136/451(30.16%), blood group 'B' is 79/451(17.51%), blood group 'AB' is 55/451(12.20%). Thus the incidence of blood group 'O' in population of Khammam is higher than other blood groups. The frequency of ABO blood groups in HIV infected individuals as compared to that of controls is as follows; blood group 'O' is 369/912(40.46 %), blood group 'A' is 286/912(31.36%), blood group 'B' is 211/912(23.14 %), blood group 'AB' is 46/912(5.04%). From this it is noticed that the frequency of blood group 'B' is increased by 5.63% as compared to that of controls (otherwise, the frequency would have been the same as in controls), blood group 'A' is increased by 1.2%, blood group 'O' there is hardly any difference, and it is decreased by 7.16% in blood group 'AB' (which may be a compensatory fall of percentage since there is a rise of the incidence in blood group 'B', 'A' & 'O').

Therefore, it appears that HIV infected individuals are more common in blood group 'B' individuals than any other blood groups and which is statistically significant (df = 3 ; X² = 25.30; P < 0.001) i.e. out of 100 only one percent might have increased by a chance. From this we can conclude that blood group 'B' individuals are more prone to develop/susceptible to HIV infection and blood group 'AB' individuals are least affected we said that the relative liability of AB individuals if taken to be one then B individuals are 3 times more susceptible to the infection and the liability is calculated by the formula ((No. of HIV infected in blood group 'B'/No. of blood group 'B' in controls) X (No. of blood group 'AB' in controls/No. of HIV infected in blood group 'AB')).

We also evaluated the incidence in Rh blood group (Table no.2), The incidence of Rh Positive and Rh Negative blood groups in controls were found to be 423/451 (93.7%) and 28/451 (6.20%) as compared to HIV infected individuals as 859/912 (94.1%) and 53/912 (5.81%) respectively. Thus, HIV infection is not related to inheritance of Rhesus factor (df= 1, X² =0.085; p = 0.7706), it is not statistically significant.

Table 1: Distribution of ABO blood groups in Controls (451) and in HIV infected individuals (912)

Controls			HIV		
Blood group	Total No. of Subjects	Percentage	Blood group	Total No. of Subjects	Percentage
O	181	40.13	O	369	40.46
A	136	30.16	A	286	31.36
B	79	17.51	B	211	23.14
AB	55	12.20	AB	46	5.04
Total	451	100	Total	912	100

(df = 3; $\chi^2 = 25.30$; $P < 0.001$). Statistically significant.

Table 2: Distribution of Rhesus factor (Rh)

Blood group	Controls	HIV
Rh Positive	423 (93.79%)	859 (94.18 %)
Rh Negative	28 (6.20%)	53(5.81%)
Total	451	912

(df = 1; $\chi^2 = 0.085$; $p=0.7706$). Statistically not significant

Table 3: Estimated Per-Act Probability of Acquiring HIV from an Infected Source, by Exposure act.

Type of Exposure	Risk Per 10,000 Exposures	Percentage (%)
Parenteral		
Blood Transfusion	9000[20]	90%
Needle - sharing during Injection drug use	67[21]	0.67%
Percutaneous (Needle stick)	30[22]	0.30%
Sexual		
Receptive anal Intercourse	50[23,24]	0.50%
Receptive Penile -Vaginal Intercourse	10[23,24,25]	0.10%
Insertive anal intercourse	6.5[23,24]	0.07%
Insertive Penile -Vaginal Intercourse	5[23,24]	0.05%
Receptive Oral intercourse	Low[23]	Low
Insertive oral intercourse	Low[23]	Low
Others		
Biting	Negligible	Negligible[26]
Spitting	Negligible	Negligible
Throwing Body fluids (Including Semen or saliva)	Negligible	Negligible
Sharing Sex Toys	Negligible	Negligible

Discussion

As we were referring literature on HIV infection on Public domains we came across this literature please see table no.3 [20], that all that are exposed to HIV are not infected and that the infection depends on number of exposure, port of entry, type of sexual activity and viral dose. In case of 10000 individuals who have been transfused an infected blood, 10% of them are not infected and the percentage of infection is even less in sexual transmission.

Therefore, it appears that something is protecting the infection. The first thing one can think of is that the body defense and thereby the genetic constitution may be providing the protection. And therefore, we thought of getting into depth of this and it is our first venture to find if the infection is related to blood groups. And therefore, we started this project and we found that blood group B individuals are more susceptible and blood group AB is least susceptible to the infection. The relative liability of AB individuals if taken to be one then B

individuals is 3.0 times more susceptible to the infection. During the course of the study we found that others also have worked on this yet we continued to work to learn the co-relation in this geographical part since so far no one has worked. Dr SK Sayal et al., in 1996[29] also had done same work and his findings are that Blood group O are more prone to HIV infection and Blood group B are less susceptible to HIV infection. But in our work we found that B was more susceptible than Blood group O.

This may be due to fact that he has investigated only in 104 HIV individuals whereas we have conducted in 912 HIV individuals. Our findings are contrary to his findings because probably he has investigated fewer samples. Results of the current study showed that the frequency of blood group 'B' in HIV infected individuals is significantly increased by 5.63% as compared to that of controls; there is a non-significant rise of HIV individuals by 1.2% in blood group 'A'; in blood group 'O' there is hardly any difference and it is significantly decreased by 7.16% in blood group 'AB'.

The results obtained in Control group and HIV infected subjects would be almost the same if there is no relation between blood group antigen and HIV. But here we found that the percentage of rise of HIV infected individuals in the said two groups is not much except in 'B' blood group and has risen by 5.63% on control group and it is statistically significant ($df = 3$; $X^2 = 25.30$; $P < 0.001$) and therefore, we assess that 'B' blood group individuals are more prone or susceptible to HIV infection.

On the other hand, there is significant fall in the percentage by 7.16% on Controls in 'AB' blood group and therefore, we can interpret that blood group 'AB' individuals are least susceptible to HIV infection. To sum up and conclude, we say that blood group 'B' individuals are more prone to get infected by HIV and blood group 'AB' individuals are more resistant to the HIV infection. On the other hand HIV infection is not related to inheritance of Rhesus factor.

Conclusion

From the observed results we can conclude that blood group 'B' individuals are more prone to get HIV infection and blood group 'AB' individuals are more resistant to the infection. HIV infection is not related to inheritance of Rhesus factor.

There is a probability that presence or absence of these antigens in plasma and their secretion into vaginal, cervical and seminal fluid may or may not as the case may be, provide protection against HIV infection at least in some of the individuals! It could also be inferred that the severity of the disease in HIV infection is modified by the genetic constitution. However, larger study involving more number of samples may be required to come to a definite conclusion.

Acknowledgements: The authors are very grateful and owe a deep debt of gratitude to the patients and healthy controls who willingly supported us and helped by participating in this study. We are also Thankful to the Management, Mamata medical college, Khammam (Telangana) for providing us the facilities to undertake these investigations in Mamata Medical College, Mamata General Hospital & Government General Hospital. We also thank our departmental colleagues who encouraged us in every step of this project.

References

1. K V Ramana and Ratna rao. Human immunodeficiency virus disease management in highly active anti-retroviral therapy era: a comprehensive review. *Ann of Trop Med Public Health*. 2013; 6(1): 5-9.
2. Geldmacher C, Ngwenyama N, Schuetz A, Petrovas C, Reither K, Heeregrave EJ, et al. Preferential infection and depletion of

- Mycobacterium tuberculosis specific CD4 T cells after HIV-1 infection. *J Exp Med*. 2010; 207(13):2869-81.
3. Mahiane SG, Legeai C, Taljaard D et al. Transmission probabilities of HIV and herpes simplex virus type 2, effect of male circumcision and interaction: a longitudinal study in a township of South Africa. *AIDS*. 2009; 23(3):377-383.
4. Migueles, S.; Connors, M. Long-term non-progressive disease among untreated HIV-infected individuals: Clinical implications of understanding immune control of HIV. *JAMA*. 2010; 304(2):194-201.
5. Lawn SD. "AIDS in Africa: the impact of co-infections on the pathogenesis of HIV-1 infection". *J. Infect. Dis*. 2004; 48(1):1-12.
6. Buchbinder SP, Katz MH, Hessol NA, O'Malley PM, Holmberg SD. bLong-term HIV-1 infection without immunologic progression. *AIDS*. 1994; 8(8):1123-8.
7. Schneider MF, Gange SJ, Williams CM, Anastos K, Greenblatt RM, Kingsley L, Detels R, Munoz A. Patterns of the hazard of death after AIDS through the evolution of antiretroviral therapy: 1984-2004. *AIDS*. 2005; 19(17):2009-18.
8. Morgan D, Mahe C, Mayanja B, Okongo JM, Lubega R, Whitworth JA. HIV-1 infection in rural Africa: is there a difference in median time to AIDS and survival compared with that in industrialized countries?. *AIDS*. 2002; 16(4):597-603.
9. Centers for Disease Control (CDC) (June 1982). A cluster of Kaposi's sarcoma and Pneumocystis carinii pneumonia among homosexual male residents of Los Angeles and orange Counties, California. *MMWR Morb. Mortal. Wkly. Rep*. 1982; 31(23):305-7.
10. Weiss RA. How does HIV cause AIDS?. *Science*. 1993; 260(5112):1273-9.
11. Sulek K. Nobel Prize in 1930 for Karl Landsteiner for discovery of human blood groups. *Wiad Lek*. 1968; 1; 21(3):233.
12. Speiser P. Smekal FG. Karl Landsteiner (translation by R Rickett). Verlag Bruder Hollinek, Vienna; 1975.
13. Von Decastello A, Sturli A. Ueber die Isoagglutinine im Serum gesunder und kranker Menschen. *Munch Med Wschr*. 1902;49:1090-1095.
14. Sigmon JM. Basic principles of the ABO and Rh blood group systems for hemapheresis practitioners. *Journal of Clinical Aphaeresis*. 1992; 7(3):158-162.
15. Clarke CA, McConnell RB, Sheppard PM. ABO Secretion of blood group antigens and Peptic ulcer. *Br Med J*. 1959; 7;1(5122):603-7.

16. Tyagi SP, Tyagi GK, Pradhan S. ABO blood group in relation to cancer cervix. *Ind J Med Sc.* 1967; 21:611-615.
17. Aird I. and Bentall, H.H. A relationship between cancer of stomach and the ABO blood groups. *Brit. Med. J.* 1953; 1(4814):799-801.
18. Clendenning WE, Boyer JT. The Skin and the haemopoietic system. In: Fitzpatrick TB, Woff K, Eisen AZ, eds. *Dermatology in general medicine.* New York: Mc Graw Hill, 1971.
19. Dacie JV, Lewis SM. *Practical Physiology.* Fifth edition, Edinburgh, E.L.B.S and Churchill Livingstone. 1975.
20. Donegan E, Stuart M, Niland JC, et al., Infection with HIV - 1 among recipients of antibody- Positive blood donations. *Ann Intern Medicine.* 1990; 113(10):733-739.
21. Kaplan EH, Heimer R. A model – based estimate of HIV infectivity via needle sharing. *J Acquir Immune Defic Syndr.* 1992; 5(11): 1116-1118.
22. Bell DM, Occupational risk of HIV infection in health care workers; an overview *Am J Med.* 1997; 102(5B):9-15.
23. Varghese B, Maher JE, Peterman TA, Branson BM, Steketee RW. Reducing the risk of sexual HIV transmission: quantifying the per-act risk for HIV on the basis of choice of partner, sex, act, and Condom use. *Sex Transm Dis.* 2002; 29(1):38-43.
24. European Study Group on Heterosexual Transmission of HIV. Comparison of female to male and male to female transmission of HIV in 563 stable couples. *BMJ.* 1992; 304(6830):809-813.
25. Leynaert B, Downs AM, de Vincenzi I; European Study Group on Heterosexual Transmission of HIV. Heterosexual Transmission of HIV: variability of infectivity throughout the course of infection. *Am J Epidemiology.* 1998; 148(1):88-96.
26. Pretty LA, Anderson GS, Sweet DJ. Human bites and the risk of HIV transmission. *Am J Forensic Med Pathol.* 1999; 20(3):232-239.
27. Sayal SK et al., Study of blood groups in HIV seropositive patients. *Indian Journal of Dermatology, venereology and Leprology.* 1996; 62(5):295-297.