International Journal Dental and Medical Sciences Research
Volume 5, Issue 3, May - June 2023 pp 11-19 www.ijdmsrjournal.com ISSN: 2582-6018

"A Study On Association Of Human Leucocyte Antigens In Patient With Psoriasis Vulgaris Attending A Teritiary Care Hospital"

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Date of Submission: 01-05-2023 Date of Acceptance: 10-05-2023

ABSTRACT:

Introduction:
Psoriasis is an auto-immune disease characterized by the infiltration of inflammatory cells into the epidermis and altered keratinocyte differentiation. Genes, immune dysregulations and environmental triggers are thecritical factors in its pathogenesis.It affects almost 0.5% to 11.4% persons of any age and gender worldwide and 0.44% to 2.8% in India. Its etiology remains elusive but genetic, metabolicandimmunologic mechanisms have been implicated. A Th1 type of immune response and inflammatory cells, elevated TNF-α and other cytokines, chemokinesand growth factors are central to the inflammation in the dermal micro environment and epidermalhyper proliferation occurs secondarily.

Aim: To evaluate distribution of HLA-A,HLA-B and HLA-C alleles and hence identify the susceptible allele of psoriasis from patients attending in tertiary care hospital.

Materials and Methods: This study is cross sectional prospective study, carried out for a period of 3months, conducted at the Department of Microbiology, Government Stanley Medical College and hospital in collaboration with Department of Dermatology, Government Stanley Medical College.Twenty five adult Patients of age 18 years and above with clinical features such as scaling, itching, dry skin, erythema, bleeding and /or histopathological diagnosis ofPsoriasis vulgaris with or without arthritis attending outpatient andinpatient department were included in my study. Blood samples (10ml) were collected aseptictallyfrom the patients and controls in a test tube containing heparin.

Results:HLA-A* 2, A* 24, A*31,A*80 ,HLA-B*7 ,B*35, B*38, HLA-CW*6 and CW*7 alleles was strongly associated with psoriasis. HLA-CW*6(64%), HLA-B*38(64%) were predominant alleles in my population.

Conclusions: There are different HLA – A, B&C alleles associated with psoriasis in this study population in comparison with other ethnic studies. Patients with HLA-Cw6 also respond better to conventional treatments and some biologics despite more extensive plaques. Future research is needed to elucidate the role of HLA-Cw6 in psoriasis.

Key words: Human Leucocyte Antigen (HLA),Tumournecrosis factor(TNF),Psoriasis,Allele

I. INTRODUCTION

Psoriasis, a common chronic inflammatory disease characterized by the infiltration of inflammatory cells in to the epidermis and altered keratinocytedifferentiation. Genes,immune dysregulationsand environmental triggers are the three critical factors in its pathogenesis.[1,2] It affects almost 0.5% to 11.4% persons of any age and gender worldwide and 0.44% to 2.8% in India. It is a chronic immune mediated disorder of the skin and joints characterized by cutaneous inflammation, epidermal hyperplasia and increased risk of arthritis as well as cardiovascular morbidity.[3] Substantial evidence indicates that psoriasis is driven by abnormal interactions between the innate and adaptive immune cells, including keratinocytes, neutrophils, macrophages, dentritic cells and T cells. Its etiology remains elusive but genetic, metabolic and immunogenic mechanisms have been implicated.[4] The human leukocyte antigen system is a gene complex encoding the major histocompatibility complex proteins in human.[5] Analysis of the pattern of familial aggregation of psoriasis suggested the likelihood that several genetic loci might be involved in producing increased susceptibility.[(6)]That HLA linked loci are involved was shown by the demonstration of positive association between psoriasis and several HLA antigens and particular with B17,B13,CW6 and DR7.[7]

The hallmark of psoriasis is sustained inflammation leading to hyper proliferation of keratinocytes and dysfunctional differentiation.The

histologic findings are acanthosis (epidermal hyperplasia).

Neo vascularization is also a prominent feature.There are disturbances in the innate and adaptive cutaneous immune responses which is responsible for the development of the psoriatic inflammation. There is an activation of the innate immune system which is driven by the endogenous danger signals and cytokines which characteristically co-exists with an auto inflammatory perpetuation in some patients.The pathogenesis of psoriasis can be conceptualized into a initiation phase possibly triggered by trauma (Koebner phenomenon), infection, drugs and a maintenance phase characterized by a chronic clinical progression..In a study conducted by Flavia de Freire Cassia et al, in Brazil, the HLA-B*57 and HLA-C*06 were found to be significantly increased in patients with psoriasis. HLA-B*57 remained high in patients with history of erythroderma, hospital internment, systemic treatment and psoriatic arthritis, showing association with disease severity.[8] In a study on transethnic analysis of psoriasis susceptibility in South Asians and Europian by Philip E.stuart et al, it was found HLA-C*06 was the top ranking psoriasis locus.[4]

Umapathy et al evaluated the distribution of HLA-A and HLA-B alleles among psoriasis patient in Western India and reported a strong association of HLA-A2,B8 and B17 antigens.[9]

R Rani et al evaluated the role of HLA-B and HLA-C alleles among psoriasis patient in North india and reported a strong association of HLA CW*0602,B*5701 and B*3701.

HLA-CW*O602 was the main allele that was increased in the group of patients in north india.[10]

Anandan A et al evaluated HLA-A*02,24 and HLA-B*35 were found to strongly associated with Psoriasis among Tamil speaking ethnic population.[11]There are few studies determining the HLA phenotypic pattern in Chennai-Therefore this study was undertaken.

Aim :

To determine the prevalent HLA phenotypic pattern among patients with psoriasis vulgaris.

Objectives:

1.To determine the prevalent HLA phenotypic pattern among patients with psoriasis with or without arthritis by complement dependent cytotoxicity method.

2.To determine the HLA phenotypic pattern of healthy controls by complement dependent cytotoxicity method.

3.To compare the frequencies of HLA phenotype in patients with psoriasis vulgaris and healthy controls.

II. MATERIALS AND METHODS:

This study is cross sectional prospective study, carried out for a period of 3 months, conducted at the Department of Microbiology, Government Stanley Medical College in collaboration with Department of Dermatology, Government Stanley Medical College.

Twentyfive adult Patients of 18 years of age and above with clinical features such as scaling, itching, dry skin, erythema, bleeding and /or histopathological diagnosis of Psoriasis vulgaris with or without arthritis attending outpatient and inpatient department were included in the study.

Blood sample (10ml) were collected aseptically from patients and controls in a test tube containing heparin.

A. HLA typing by complement dependent cytotoxicity method

Separation of peripheral blood lymphocytes were done and subjected to HLA typing by complement dependent cytotoxicity method.

B. Other investigations

The Patients blood were also subjected to latex agglutination test for detection of rheumatoid factor, complete blood count.

C. Clinical history and examination

A clinical history and examination of all patients and the controls were recorded as per the proforma.

Complement dependent cytotoxicity Methods:

Lymphocyte separation:

Heparinised blood was gently mixed and diluted with 3ml of phosphate buffer saline(PBS) to make a total volume of 6ml .

3ml of lymphocyte separation medium, density 1.077 (lymphoflot,innotrain) was taken in a 15ml graduated centrifuge tube, overlaid gently with 5ml of diluted heparinised blood and centrifuged at 2000rpm for 30 minutes.

The buffy coat was carefully pipetted out from the plasma- lymphocyte separation medium (LSM) interface and transferred to another test tube.

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The cells were resuspended in about 4ml of PBS,mixed gently with a Pasteur pipette and centrifuged at 1000 rpm for 10 mins.
The supernatant was totally discarded and washing step repeated.
After washing the buffy coat twice,the lymphocyte was suspended in 200-300 microlitre of PBS and counted in RBC square of a Neubauer counting chamber to attain a concentration of 2*10 /ml.
The viability was checked by added by adding 1%trypanblue. viable cells were unstained while dead cells were stained.[12]

HLA TYPING based on Complement dependant cytotoxicity method:

To the Terasaki plate containing 72 HLA antibody coated wells,1microlitre of the lymphocytes are added. Positive and negative controls are also added to the appropriate wells.

Then 5microlitre of thawed complement is added and incubated at room temperature for 60 minutes and add 5microlitre of 4% eosin dye and after five minutes formol saline is added to fix the reaction.
The Plate is read after 30 minutes.[12]

Inclusion criteria:

Twentyfive adult Patients of 18 years of age and above with clinical features such as scaling, itching, dry skin, erythema, bleeding and /or histopathological diagnosis of Psoriasis vulgaris with or without arthritis attending outpatient and inpatient department was included in the study.
Twentyfive adult patient age and gender matched healthy control was also included.

Exclusion criteria:

Patients with other autoimmune disorder and malignancy were excluded in this study.
Pregnant and lactating women is excluded in the study.

PASI (psoriasis Area and Severity Index) Score:

This was developed by Fredricksson and Petterson in 1978. Both intensity and extent of the Psoriatic plates are calculated separately for four anatomical regions (head, trunk, upper and lower extremities) by the dermatologist. The intensity of erythema, desquamation and induration is rated on a 5 point scale with 0 - no involvement, 1- slight, 2- Moderate, 3-Severe, 4-Very Severe characteristics. The percentage of involvement of the four anatomical regions is assigned a numerical value of 0-6 with 0– no involvement, 1- 1 to 9%, 2- 10 to 29%, 3 –30 to 49%, 4–50 to 69%, 5–70 to 89%, 6–90 to 100% of body surface area involvement (BSA). When calculating the PASI score four anatomical regions are calculated according to the proportion of the whole involvement of the skin. The PASI score varies from 0 to 72. Higher score indicates severe conditions. The main advantage of PASI score is that it is the gold standard in clinical trials.[17]

Statistical Analysis:

All the data was entered in excel sheet and association between HLA
and psoriasis was done by Statistical analysis by chi square test using spss/version-16.

III. RESULTS:

This study is cross sectional prospective study, carried out for a period of 3month,at the Department of Microbiology, Government Stanley Medical College and hospital in collaboration with Department of Dermatology, Government Stanley Medical College. Twentyfive adult Patients of 18 years of age and above with clinical features such as scaling, itching, dry skin, erythema, bleeding and /or histopathological diagnosis of Psoriasis vulgaris with or without arthritis attending outpatient and inpatient department was included in the study.

Majority of patients who suffered from psoriasis vulgaris ranged between 30 to 70 years old. In my study male was predominant than female, most of the patients the duration of psoriasis is 5-10 years.

In this study the frequency of all allelic group of HLA-A,B,C is compared with appropriate controls. Among this HLA-A* 2, A* 24, A*31,A*80 ,HLA-B*7 ,B*35, B*38,HLA-BW*4, BW*6, HLA-CW*6 and CW*7 alleles is strongly associated with psoriasis. In that HLA-B*38, HLA-CW*6 was most commonly associated with psoriasis which is compared with various populations worldwide.
Table 1. described the gender of patients with appropriate controls.
Table 2. Describes the age of patients with appropriate controls.
Table 3. Describes the duration of patients suffering from psoriasis vulgaris.
Table 4. Describes the severity of PASI Score with mild, moderate and severe.
Table 5. Describes the individual alleles Frequency of HLA-A Allelic Groups in Patients with appropriate Controls. In this study the most predominant alleles associated with psoriasis are HLA-A*80, HLA-A*31, HLA-A*2 in order of decreasing frequency. HLA-A*1 have a negative association with psoriasis in my population.

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Volume 5, Issue 3, May - June 2023 pp 11-19 www.ijdmsrjournal.com ISSN: 2582-6018

Table 6. Describes Frequency of HLA-B Allelic Groups in Patients with appropriate Controls. In this study, the predominant HLA-B Alleles associated with psoriasis are HLA-BW*7 and HLA-B*38. HLA-B*35 have a negative association with psoriasis in my population.

Table 7. Describes theFrequency of HLA-CW Allelic Groups in Patients with appropriate Controls. In this study,the predominant HLA- C Alleles associated with psoriasis are HLA-CW*6 and HLA-CW*7.
HLA-CW*4 have a negative association with psoriasis in my population.

Table 8. Describes the HLA -A,B&C Allelic Combination Results. In this study HLA-A*31,HLA-B *38,HLA-CW*6 combined alleles was observed in 28% of cases studied and HLA–A*2, A*24, B*38, CW*6 alleles combination was observed in 24% of cases studied.

Base line characteristics of psoriasis patients and controls

1. Gender of patients and controls:

Gender	No of patients(n=25)	No of controls(n=25)
Male	16(64%)	16(64%)
Female	9(36%)	9(36%)

In this study, male was predominant than female in psoriasis patients (64%).

2. Age of patients and controls:

Age in years	No of patients(n=25)	No of controls(n=25)
<20	1(4%)	1(4%)
21-30	1(4%)	1(4%)
31-40	6(24%)	6(24%)
41-50	7(28%)	7(28%)
51-60	5(20%)	5(20%)
>60	5(20%)	5(20%)

The incidence of psoriasis is high among the age group of 40 to 50 years (28%).

3. Duration of psoriasis:

Duration of Psoriasis	No of patients(n=25)
< 5yrs	6(24%)
5-10yrs	10(40%)
>10yrs	9(36%)

In this study, around 40 % of patients are in duration of 5-10 years of disease

4. PASI score severity of patients with psoriasis:

PASI Score (Severity)	No of patients (n=25)
<6 (Mild)	9(36%)

6-12(Moderate)	11(44%)
>12 (Severe)	5(20%)

In this study 44 % of majority of patients was seen with moderate PASI score(6-12)

5. Frequency of HLA-A Allelic Groups in Patients and Controls

HLA-A*	Psoriasis (n= 25)	Controls (n=25)	P	OR(CI)
HLA-A* 1	**4(16%)**	**8(32%)**	**0.185**	**0.071(0.034-9. 625)**
HLA-A* 2	**7(28%)**	**4(16%)**	**0.306**	**2.290(0.123-2.948)**
HLA-A* 3	2(8%)	3(12%)	0. 637	1. 658(0.239-10.30)
HLA-A* 11	4(16%)	5(20%)	1.0	1(0.059- (16.92)
HLA-A* 23	1(4%)	1(4%)	1.0	1(0.059- (16.92)
HLA-A* 24	6(24%)	3(12%)	0.269	0.432(0.095-1.96)
HLA-A* 26	2(8%)	1(4%)	0. 552	0.479(0.041-5. 652)
HLA-A* 28	2(8%)	2(8%)	1.0	1(0.130-7.717)
HLA-A* 31	**7(28%)**	**2(8%)**	**0.06**	**2.224(0.041-2.210)**
HLA-A* 32	2(8%)	3(12%)	0. 637	1.568(0.239-10.30)
HLA-A* 33	3(12%)	0	0.074	0.468(0.345-0. 635)
HLA-A* 74	1(4%)	0	0.312	0.490(0.368-0. 652)
HLA-A* 80	**9(36%)**	**0**	**0.003**	**2.685(0.352-10.45**

In this study, the most predominant alleles associated with psoriasis are HLA-A* 80, HLA-A* 2,HLA-A* 31, in order of decreasing frequency.

6. Frequency of HLA-B Allelic Groups in Patients and Controls:

HLA-B*	Psoriasis (n=25)	Controls (n=25)	P	OR(CI)
HLA-BW* 4	21(84%)	18(72%)	0.306	0.490(0.123-1.948)
HLA-B* 5	2(8%)	0	0.149	0.479(0.357-0. 644)
HLA-BW* 6	19(76%)	16(64%)	0.355	0. 561(0.164-1.918)
HLA-B* 7	8(32%)	2(8%)	0.0034	0.185(0.035-0.983)
HLA-B* 8	3(12%)	2(8%)	0. 637	0. 638(0.097-4.188)
HLA-B* 15	4(16%)	3(12%)	0. 684	0.716(0.143-3. 589)
HLA-B* 18	3(12%)	0	0.074	0.468(0.345-0. 635)
HLA-B* 21	1(4%)	0	0.314	0.490(0.368-0. 652)
HLA-B* 27	2(8%)	0	0.149	0.479(0.357-0. 644)

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Volume 5, Issue 3, May - June 2023 pp 11-19 www.ijdmsrjournal.com ISSN: 2582-6018

HLA-B* 35	**5(20%)**	**7(28%)**	**0. 508**	**0. 1556(0.119-5.779)**
HLA-B* 37	3(12%)	0	0.203	0. 582(0.386-0. 686)
HLA-B* 38	**16(64%)**	**2(8%)**	**0.01**	**3.049(0.009-3.257)**
HLA-B* 39	2(8%)	1(4%)	0. 552	0.479(0.041-5.652)
HLA-B* 40	5(20%)	2(8%)	0.221	0.348(0.061-1.993)
HLA-B* 48	4(16%)	1(4%)	0.157	0.219(0.023-2.114)
HLA-B* 51	3(12%)	1(4%)	1.0	1.0(0.130-7.717)
HLA-B* 52	3(12%)	0	0.074	0.468(0.345-0. 635)
HLA-B* 55	2(8%)	0	0.149	0.479(0.0357-0. 644)
HLA-B* 57	2(8%)	2(8%)	1.0	1,0(0.130-7.717)
HLA-B* 58	3(12%)	0	0.074	0.468(0.345-0. 635)
HLA-B* 63	3(12%)	1(4%)	0.074	0.468(0.345-0.635)
HLA-B* 70	2(8%)	0	0.149	0.479(0.357-0. 644)

In this study, the predominant HLA- B associated with psoriasis are HLA-B*38. HLA-BW*4 and HLA-BW*6 is seen in both psoriatic and healthy individuals

7. Frequency of HLA-CW Allelic Groups in Patients and Controls:

HLA-CW	**Psoriasis (n=25)**	**Controls (n=25)**	**P**	**OR**
HLA-CW*1	2(8%)	1(4%)	0.552	0.479(0.041-5.652)
HLA-CW*3	4(16%)	2(8%)	0.384	0.457(0.76-2.75)
HLA-CW*4	**2(8%)**	**10(40%)**	0.008	1.470(0.91-27.21)
HLA-CW*6	16(64%)	8(32%)	0.024	2.265(0.082-2.854)
HLA-CW*7	14(56%)	5(20%)	0.009	0.196(0.056-0. 619)

In thisstudy,the predominant HLA- CW allele associated with psoriasis are HLA-CW*6

8.HLA -A & B Allelic Combination Results(Statistically Significant Findings)

Allele combination	**Total cases(N=25)**	**Total controls**	**Male**	**Female**
HLA–A*31, B *38, CW*6	7(28%)	0	6	1
HLA–A*2, A*24, B*38, CW*6	6(24%)	0	5	1

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Volume 5, Issue 3, May - June 2023 pp 11-19 www.ijdmsrjournal.com ISSN: 2582-6018

In this study HLA-A*31,HLA-B*38,HLA-CW*6 combined alleles was observed in 28% of cases studied and HLA–A*2, A*24, B*38, CW*6 alleles combination was observed in 24% of cases studied.

IV. DISCUSSION

The present study was undertaken to determine the prevalent of HLA phenotypic pattern among patients with psoriasis vulgaris and healthy controls with or without arthritis by complement dependent cytotoxicity method and to compare the frequencies of HLA phenotype in patients with psoriasis vulgaris and healthy controls.Blood sample (10ml) is collected aseptically from twenty five patients with Psoriasis and healthy controls in a test tube containing heparin attending the institute of Stanley medical college.

The present study was carried out in the Department of Microbiology, Government Stanley Medical College and hospital in collaboration with Department of Dermatology, Government Stanley Medical College.

In this study, HLA alleles associated with psoriasis are identified and compared with normal healthy population..

In this study, the most predominant alleles in HLA-Aassociated with psoriasis is HLA-A*80, HLA-A*2, HLA-A*31, in order of decreasing frequency.

HLA-A*80 with odds of **2.685(0.352-10.45)with p**-value of **0.003,**HLA-A*2 with odds of **2.290(0.123-2.948) with p**-value of **0.306,and** HLA-A*31with odds of **2.224(0.041-2.210)with p**-value of **0.06.**All these 3 alleles are statistically significant and shows strongly associated with Psoriasis.

Among the control groups in HLA A*1 with odds of **0.071(0.034-9. 625) with p**-value of **0.185 is negatively associated with psoriasis. (TABLE 5**)

In this study the most predominant alleles in HLA-B associated with psoriasis is HLA-B*38 with odds of **3.049(0.009-3.257) with p**-value of **0.01 is strongly associated with psoriasis** was statistically significant.

HLA-B*35 with odds of **0. 1556(0.119-5.779)with p**-value of **0. 508 is negatively associated with psoriasis. (TABLE6**).

In this study, the most predominant alleles in HLA-CW associated with psoriasis is HLA-CW6 with odds of 2.265(0.082-2.854) **with p**-value of 0.024 **is strongly associated with psoriasis and** statistically significant.

HLA-CW4 is negatively associated with psoriasis**(table 7).**

HLA-CW4 with odds of 1.470(0.91-27.21) with **p**-value of 0.008 is negatively associated with psoriasis.

In this study HLA-A*31,HLA-B*38,HLA-CW*6 combined alleles was observed in 28% of cases studied and HLA–A*2, A*24, B*38, CW*6 alleles combination was observed in 24% of cases studied.

In many studies shows HLA-CW6 has been proven to strongly associated with psoriasis from various ethnic population worldwide by Ashwin Anandan et al[11] (2020).SchmittEgenolf et al and choonhakam et al reported similar haplotypes in german and thai patients with psoriasis

In that HLA-A*80 ,HLA-B*38, and HLA-CW6 is most commonly associated with psoriasis.Similar studies shown by J C Woodrow et al[6], Nair RP et al[15]

HLA-A*2, A* 80, B*38, HLA-CW*6 are the ones that particularly carry genes involved in susceptibility to psoriasis. Similar studies shown by Ashwin Anandan et al[11] (2020)

HLA-CW*6 and HLA-B*38 has been proven to be strongly associated with psoriasis from various population worldwide. There are various studies shows being strongly associated with psoriasis.[15]

Russel et al first reported in 1972, that HLA-B*13 and HLA-B*17 to be associated with psoriasis[16] ,Chablani et al showed HLA-B*17 association with psoriasis.[17]

Comparing these findings with my current study, there are certain other HLA alleles also to be considered having association with psoriasis.

HLA-A*2, HLA-A*24, HLA-A*31, HLA-B*18,HLA-B*7,B*38 are some of the alleles was positive association with psoriasis vulgaris and psoriatic arthritis also[11].

HLA-A*01 HLA-B*35 andHLA-CW*04 were found in increased frequency among the controls, compared to psoriasis patients, implying these alleles may be protective against the disease. These alleles are protective alleles was previously described as a Protective antigens by Flavia de Freire et al.[8]

In this study it is observed that HLA-BW*4, HLA-BW6 was seen associated with both psoriatic and healthy individuals.

V. SUMMARY:

This study is cross sectional prospective study was carried out for a period of 3month at the Department of Microbiology, Government Stanley Medical College and hospital in collaboration with

Department of Dermatology, Government Stanley Medical College. Twentyfive adult patients age and gender matched healthy control was included in this study.

Among the 25 adult patients with Psoriasis, the incidence of Psoriasis was found predominantly among male patients(64%) than female.

Among the 25 adult patients with Psoriasis,the incidence of Psoriasis was high among the age group of 40 to 50 years (28%). In this study majority of patients was seen in moderate PASI score with average duration of disease ranging from 5 to 10 years. (64%).

Among the 25 adult patients with Psoriasis in that HLA-A*80**(36%)**, HLA-B*38**(64%)**and HLA-CW*6**(64%)** was the predominant alleles was observed in my study.These alleles have a positive association with psoriasis,whereas HLA-A*01**(32%)** ,HLA-B* 35**(28%)**andHLA-CW*04(40%) have a negative association with Psoriasis in my population.

In this study HLA- A*01 HLA- B* 35 andHLA-CW*04 may have a protective effect against the disease. In this study HLA-A*31,HLA-B *38,HLA-CW*6 combined alleles was observed in 28% of cases studied and HLA–A*2, A*24, B*38, CW*6 alleles combination was observed in 24% of cases studied.

In this study HLA-B*38(64%) and HLA-CW*6(64%), was the predominant alleles found in my population and most commonly associated with Psoriasis vulgaris.

It is observed that HLA-BW*4, HLA-BW6 was seen in high frequency with both groups(psoriatic and healthy individuals).

VI. CONCLUSIONS:

In this study twentyfive adult patients with Psoriasis were admitted and healthy controls as outpatient conducted at the Department of Microbiology, Government Stanley Medical College and hospital in collaboration with Department of Dermatology was studied during the period of 3 month.

In this study the incidence of Psoriasis was found predominantly among male patients(64%) than female.The incidence of Psoriasis is high among the age group of 40 to 50 years (28%) and majority of patients was seen in moderate PASI score with average duration of disease ranging from 5 to 10 years(64%). HLA-A*80**(36%)**, HLA-B*38**(64%)** and HLA-CW*6**(64%)** was observed in my study is the most commonly associated with Psoriasis vulgaris. In our Population the results showed that HLA-B*38(64%) and HLA-CW*6(64%) was the main allele that was increased in this group of patients. HLA-A*01**(32%)** ,HLA-B* 35**(28%)**andHLA-CW*04(40%) have a negative association with Psoriasis vulgaris. It may have a protective effect against the disease. However,to come to this conclusion on the basis of the results from the sample size of the present study, HLA-B*38**(64%)** and HLA-CW*6**(64%) was the predominant alleles**,thereby act as main effector cells in maintaining the pathogenic process and major susceptibility risk allele for psoriasis. Human leukocyte antigen (HLA)-Cw6 is the disease allele conferring the greatest risk to psoriasis, but its prevalence is lower in Asian individuals. Patients with HLA-Cw6 also respond better to conventional treatments and some biologics despite more extensive plaques.Further research need to elucidate the role of these alleles.

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