HLA-B and HLA-C Alleles in Patients with Psoriasis, Psoriatic Arthritis and Undifferentiated Spondyloarthritis in a Tertiary Care Hospital of Bangladesh

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Abstract:

Background:
The frequency of HLA-B and HLA-C alleles differs among psoriasis, psoriatic arthritis, and undifferentiated spondyloarthritis, showing unique genetic risks. These genetic links demonstrate how MHC class I alleles affect immune issues, influencing clinical classification and research for specific treatments.

Objective:
To determine the frequency of HLA-B and HLA-C alleles in patients with psoriasis, psoriatic arthritis (PsA), and psoriatic phenotype of undifferentiated spondyloarthritis (uSpA), with clinical features suggestive of psoriatic arthritis but do not fulfill the CASPAR criteria.

Methods:
The cross-sectional study involved 29 patients with PsA, 23 with uSpA, 33 with psoriasis, and 30 healthy controls. Symptoms in uSpA patients suggest PsA, including dactylitis, enthesitis, back pain, DIP involvement, and FADES-like skin changes. HLA-B and HLA-C alleles were detected.

Results:
In PsA patients HLA-B*27 allele was detected in 9 (31.03%, p=0.67); HLA-B*15 in 9 (31.03%, p=0.49), HLA-B*38 in 2 (4.5%, p=0.20) and HLA-C*8 in 10 (34.5%, p=0.02). In uSpAfrequency of HLA-B*38 (N=15, 65.21%, p=0.07) and HLA-C*4 alleles (N=8, 34.8%, p=0.20) were more frequent than the healthy controls. Among uSpA patients with DIP involvement, the frequency of HLA-B*38 (N=7, 43.8%, p=0.01) was increased and those who had FADES like skin lesions, HLA-B*38 (N=5, 45.5%, p=0.999) was also increased. Increased frequency of HLA-B*57 was found in psoriatic patients (N=13, 39.4%, p=0.52).

Conclusion:
The prevalence of HLA-B*27 and HLA-C*8 is notably elevated in patients with psoriatic arthritis (PsA), whereas HLA-B*38 is found at a lower frequency. In the case of undifferentiated spondyloarthritis (uSpA), individuals exhibiting distal interphalangeal (DIP) joint involvement and presenting with FADES-like skin lesions may show a higher occurrence of HLA-B*38. This observation implies that some patients currently classified as uSpA according to existing criteria might have psoriatic arthritis.

Keywords: Psoriasis, Psoriatic arthritis, Undifferentiated spondyloarthritis, FADES, HLA alleles

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Introduction:

Psoriasis is a common skin disorder, with a prevalenceof 0% to 11.8% in different population.[1] Association with HLA-A*01, HLA-A*02, HLA-B*13, HLA-B*17, HLA-B*39, HLA-B*57 variesamong different racial and ethnic backgrounds.[2] Psoriatic arthritis (PsA) is a heterogeneous disease with varying clinical and radiographic manifestations.[3]

About 10-42% of patients with psoriasis may develop PsA.[4] Genetic, environmental, and immunological factors might play a role in development and perpetuation of the disease, though the pathogenesis is not clear. Class I HLA antigens, encoded by HLA-A, B, and C loci of the major histocompatibility complex have been associated with psoriatic arthritis.[5] Patients withSpA may be classified according to European Spondyloarthropathy Study Group (ESSG) criteria or Amor criteria previously.[6,7] However, the Assessment of Spondyloarthritis International Society (ASAS) group had developed a new set of criteria to differentiate axial or peripheral SpA.According to ASAS it is to consider all SpA patients with predominantly axial involvement as axial SpA irrespective of whether they have definite radiographic sacroiliitis.Peripheral SpAs are subdivided into reactive arthritis (ReA), PsA and arthritis associated with inflammatory bowel disease.[8,9] PsA is diagnosed based on presence of psoriasis skin and nail lesions in the patient or in his/her family. Many physicians apply CASPAR criteria in clinical practice to diagnose psoriatic arthritis.[10] The patients were diagnosed as uSpA, when they do not fulfill any of the above categories.Frictional asymptomatic darkening of the extensor surfaces (FADES), also known as hyperkeratosis of the elbows and knees, has never been well characterized. The patients present with uniform, asymptomatic, brown darkening over the extensor surfaces of the elbows and knees with minimal scaling.[11] Rheumatoid factor can be positive in 2 to 10% of patients with psoriatic arthritis. Anti- cyclic citrullinated peptide antibody (ACPA) can be positive in 12.2% of patients with PsA but at low titer.[12,13]

This study primarily aimed to identify HLA associations of psoriasis and PsA in the Bangladeshi community. We took the opportunity of studying some of our uSpA labeled patients in reality or genetically may have PsA. In our clinical practice, some of the subjects classified as RA as per ACR/EULAR 2010 criteria and not meeting the CASPAR criteria are also found to have some distinctive features of PsA (e.g. DIP involvement, dactylitis, enthesitis, inflammatory back pain and FADES like skin changes).As a tertiary objective, we tried to study if some of these patients may have psoriasis or PsA alleles.

Methods:

This cross-sectional study was carried out in outpatient department of Rheumatology, and Dermatology, and department of Microbiology, BSM Medical University, Dhaka, Bangladesh, over a period of 2 years March 2015 to April 2017. Patients with PsA (according to CASPAR criteria), uSpA (according to ESSG criteria) with psoriatic arthritis phenotype, uSpA with FADES like skin lesions and rheumatoid arthritis satisfying ACR-EULAR-2010 criteria but with some pointers to PsA were consecutively enrolled in the study. SpA patients who did not fulfill criteria for ankylosing spondylitis, reactive arthritis, PsA, arthritis associated with IBD were categorized as uSpA. Patients of uSpA who had clinical features suggestive of psoriatic arthritis like enthesitis and/or dactylitis and/or distal interphalangeal joint involvement and/or subungual hyperkeratosis but did not fulfill CASPAR criteria of PsA were enrolled. The patients who had inflammatory bowel disease (IBD), psoriasis, a preceding symptomatic infection of the urogenital or the gastrointestinal tract within the 4 weeks before the onset of SpA symptomswere excluded. Patients with psoriasis were diagnosed clinically by a dermatologist. The control subjects were recruited from postgraduate medical students, nursing staffs and employees working in outpatient department of BSM Medical University. The study involved control subjects without psoriasis or no family history of psoriasis and SpA. Human Leukocyte Antigen (HLA) tissue typing was performed using The MorganTM HLA SSP ABDR typing Kit. Ethical approval was obtained from BSM Medical University. Data analysis compared HLA-B and HLA-C allele frequencies in patient groups with control subjects using chi-square test and Fisher's exact test, p-value<0.05 were significant.

Results:

In this study 29 psoriatic arthritis patients, 23 undifferentiated spondyloarthritis patients, 33 psoriatic patients and 30 healthy subjects were included. Out of 23 undifferentiated spondyloarthritis patients with clinical features suggestive of psoriatic arthritis, 4 had positive RF or ACPA.

Mean age of the patients with PsA, uSpA, psoriasis and healthy controls were respectively 44.72±10.84, 44.13±13.48, 37.48±10.84 and 31.07±7.66 years. Female were predominant in

PsA and uSpA in contrast male in psoriasis and healthy controls (Table-I).

Table-I: Age and sex distribution of the participants (N=115)

Demographics	PsA (n=29)	uSpA (n=23)	Psoriasis (n=33)	healthy controls (n=30)
Mean age (±SD) in years	44.72±10.84	44.13±13.48	37.48±10.84	31.07±7.66.
Male no. (%)	11(37.9)	7(30.4)	23(69.7)	19(63.3)
Female no. (%)	18(62.1)	16 (69.6)	10(30.3)	11(36.7)

Polyarticular involvement was most common in both PsA and uSpA. Inflammatory back pain was more in PsA and enthesitis in uSpA (Table-II).

DIP-Distal interphalangeal joint; FADES**:** Frictional asymptomatic darkening of extensor surfaces

HLA-B*15 and HLA-B*27 were increased in psoriatic arthritis. We have also found that HLA-B*27, HLA-B*38 and HLA-B*57 were detected more frequently in patients with undifferentiated spondyloarthritis. HLA-C*4, HLA-C*7were found in increased frequency in patients with psoriatic, psoriatic arthritis and uSpA. (Table-III and IV)

Table-II: Clinical features of PsA and uSpA

Clinical feature	PsA (n=29) no. (%)	uSpA (n=23) no. (%)
Duration of Arthritis in months (Mean±SD)	72.10±68.58	44.57±50.25
Polyarticular	24(82.8)	16(69.6)
Oligoarticular	5(17.2)	7(30.4)
Monoarticular	0(0.0)	0(0.0)
Inflammatory back pain (IBP)	16(55.2)	9(39.1)
Enthesitis present	7(24.1)	9(39.1)
DIP involvement	18(62.1)	16(69.6)
FADES	-	11 (47.82)

Table-III Frequency of HLA-B alleles in disease groups (n=85) and controls (n=30)

Alleles	Controls (n=30) no. (%)	Psoriatic arthritis (n=29)			uSpA (n=23)			Psoriasis (n=33)		
		no. (%)	RR	p-value	no. (%)	RR	p-value	no. (%)	RR	p-value
B*7	1(3.3)	1(3.4)	1.02	0.98	3(13.0)	1.84	0.07	3(9.1)	1.48	0.22
B*13	1(3.3)	2(6.9)	1.38	0.46	3(13.0)	1.84	0.07	3(9.1)	1.48	0.22
B*15	7(23.3)	9(31.0)	1.21	0.49	4(17.4)	0.80	0.62	5(15.2)	0.76	0.45
B*17	1(3.3)	0(0.0)	-	-	0(0.0)	-	-	0(0.0)	-	-
B*27	5(16.7)	9(31.0)	1.10	0.67	6(26.08)	0.90	0.73	1(3.0)	0.30	0.19
B*35	6(20.0)	3(10.3)	0.64	0.37	1(4.3)	0.30	0.20	7(21.2)	1.04	0.90
B*37	3(10.0)	3(10.3)	10.2	0.96	0(0.0)	-	-	4(12.1)	1.10	0.78
B*38	5(16.7)	2(4.5)	0.46	0.20	15(65.21)	1.40	0.07	2(6.1)	0.52	0.28
B*39	0(0.0)	0(0.0)	-	-	1(2.3)	1.70	<0.001	1(3.0)	1.94	<0.001
B*44	5(16.7)	4(13.8)	0.89	0.77	5(21.7)	1.19	0.63	6(18.2)	1.05	0.87
B*51	0(0.0)	3(10.3)	2.15	<0.001	1(4.3)	2.36	<0.001	1(3.0)	1.94	<0.001
B*57	7(23.3)	4(13.8)	0.70	0.40	4(17.4)	0.80	0.62	13(39.4)	1.40	0.52

chi-square test

Table-IV: Frequency of HLA-C alleles in disease groups (n=85) and in healthy controls (n=30)

Allele	Controls (n=30) no. (%)	Psoriatic arthritis (n=29)			uSpA (n=23)			Psoriasis (n=33)		
		no. (%)	RR	p-value	no. (%)	RR	p-value	no. (%)	RR	p-value
C1	4(13.3)	2(6.9)	0.65	0.48	1(4.3)	0.43	0.37	0(0.0)	-	-
C2	3(10.0)	3(10.3)	1.02	0.96	0(0.0)	-	-	0(0.0)	-	-
C3	3(10.0)	0(0.0)	-	-	0(0.0)	-	-	1(3.0)	0.46	0.38
C4	6(20.0)	2(6.9)	0.47	0.24	8(34.8)	1.49	0.20	9(27.3)	1.20	0.48
C5	3(10.0)	0(0.0)	-	-	1(4.3)	0.56	0.51	1(3.0)	0.46	0.38
C6	5(16.7)	4(15.9)	0.98	0.08	4(9.1)	0.72	0.40	6(18.2)	1.05	0.87
C7	12(40.0)	10(34.5)	0.88	0.67	9(39.1)	0.98	0.95	16(48.5)	0.18	0.50
C8	3(10.0)	9(31.0)	1.76	0.02	3(13.0)	1.18	0.72	3(9.1)	0.95	0.90
C9	0(0.0)	3(10.3)	2.15	<0.001	0(0.0)	-	-	0(0.0)	-	-
10	2(6.7)	2(6.9)	1.02	0.04	00.0()	-	-	2(6.1)	0.95	0.92

chi-square test

HLA-B38 and HLA-B44 were more frequent inpatients with uSpA with DIP involvement. In case of PsA with DIP joint involvement HLA-B15, HLA-B44, and HLA-B57 detected more frequently (Table-V).

Table-V: Frequency of HLA-B alleles on psoriatic patients (n=18) and patients with uSpA with distal interphalangeal joint involvement (n=16)

Alleles	PsA (n=18) no. (%)	uSpA (n=16) no. (%)
HLA-B*7	1(5.6)	2(12.5)
HLA-B*13	0(0.0)	2(12.5)
HLA-B*15	5(27.8)	2(12.5)
HLA-B*17	0(0.0)	0(0.00)
HLA-B*27	1(5.6)	0(0.00)
HLA-B*35	3(16.7)	1(6.3)
HLA-B*37	2(11.1)	0(0.0)
HLA-B*38	1(5.6)	7(43.8)
HLA-B*39	0(0.0)	0(0.00)
HLA-B*44	4(22.2)	3(18.8)
HLA-B*51	2(11.1)	1(6.3)
HLA-B*57	4(22.2)	2(12.5)

HLA-B*38 was more frequentamong the patients with uSpA with FADES like skin lesions (Table-VI).

Table-VI: Frequency of HLA-B alleles on uSpA with and without FADES like skin lesions (n=11)

Alleles	FADES like skin lesions	
	Present (n=11) no. (%)	Absent (n=12) no. (%)
HLA-B*7	2(18.2)	1(8.3)
HLA-B*13	1(9.1)	2(16.7)
HLA-B*15	3(27.3)	1(8.3)
HLA-B*17	0(0.0)	0(0.0)
HLA-B*27	1(9.1)	0 (0.0)
HLA-B*35	0(0.0)	1 (8.3)
HLA-B*37	0(0.0)	0 (0.0)
HLA-B*38	5(45.5)	5 (41.7)
HLA-B*39	0(0.0)	0 (0.0)
HLA-B*44	2(18.2)	3 (25.0)
HLA-B*51	1(9.1)	0 (0.0)
HLA-B*57	1(9.1)	3(25.0)

FADES: Frictional asymptomatic darkening of extensor surfaces

Discussion:

In our study the remarkable finding is that there is an increased frequency of HLA-B*13, HLA-B*35, HLA-B*37, HLA-B*44, HLA-C*04, HLA-C*06 and HLA-C*07 in psoriatic patients. HLA-B*13, B*35, B*37 had a relative risk of 1.48, 1.04 and 1.10 respectively. HLA-B*15, HLA-B*27 and HLA-B*38 showed a decreased association with psoriasis with relative risk of 0.76, 0.30 and 0.52

respectively. HLA-C*04, C*06 and C*07 have a frequency of 27.3%, 18.2% and 48.5% respectively. HLA-C*04 have a relative risk of 1.20 and HLA-C*06 have a relative risk of 1.05.

Russel et al in 1972 reported an association of HLA-B*13 and HLA-B*17 with psoriasis vulgaris.[14] He also showed that the association of HLA-C*16 was stronger than HLA-B*13 and HLA-B*17. A study from Northern India showed that HLA-B*17 to be associated with psoriasis but HLA-B*13 was not found in that study.[15] Ashwin A et al found that HLA-B*40 and HLA-B*18 had an increased association with psoriasis and HLA-B*35 was also found in a higher frequency than the controls. He also reported that HLA-B*17 was not found in his study and HLA-B*13 was found in a lesser frequency.[16] In our study HLA-B*13 was found in 3 out of 30 patients (9.1%) with a relative risk of 1.48 and in 1 out of 30 (6.9%) controls. Our study also showed HLA-B*13 to be found in increased frequency in cases when compared with the controls. HLA-B*17 was not found in our study.Rani R et al showed that the frequency of C*06 was 30% in North India.[17] Ozawa A et al reported that there is increased frequency of C locus antigens such as C*02, C*04, C*07, C*11 in psoriatic populations in Japan.[18] Woodrow JC et al showed that there was positive association between HLA-C*6 allele and psoriasis. He also reported that there were increased frequencies of HLA-C*6 (72%) and HLA-C*7 (34%) in psoriatic patients.[19] We found that there was increased frequency of HLA-B*15 (31.0%), HLA-B*27 (31.0%) and HLA-B*51 (10.3%) in patients with psoriatic arthritis. In our study HLA-B*35 (10.3%) and HLA-B*38 (4.5%) were found in a lesser frequency in patients with psoriatic arthritis. HLA-B*39 was not found in our study. HLA-C*6 (15.9%), HLA-C*7 (34.5%) and HLA-C*8 (31.0%) are more frequent in psoriatic arthritis patients in our study. Woodrow JC et al showed that there was increased frequency of HLA-B*13 (18%), HLA-B*17 (32%) and HLA-B*27 (26%) in patients with psoriatic arthritis. They also reported that HLA-C*6 was more frequent in psoriatic arthritis.[19] In a study on Spanish population showed that HLA-B*17 (19.2%) and HLA-B*27 (22.1%) were common in patients with psoriatic arthritis. They also reported that HLA-C*6 (20.1%) was more frequent in patients with psoriatic arthritis.[20] In our study we have also found increased frequency of HLA-B*27 in psoriatic arthritis patients. We have found HLA-B*7 in 1 out of 29 patients with psoriatic arthritis. HLA-B*17 was not found in our study.In our study HLA-B*27 (26.08%), HLA-B*38 (65.21%) and HLA-B*44 (21.7%) were detected in increased frequency in patients with undifferentiated spondyloarthritis. HLA-C*4 and HLA-C*7 were also found more frequently in this patents group. Mishra et al. observed HLA-B*27 in 56% of patients with uSpA.[21] A study from Mexican patients with uSpA revealed that HLA-B*27 and HLA-B*15 were frequent in these patients group.[22] In our study we found increased frequency of HLA-B*27 in patients with uSpA. HLA-B*15 was found in 4 out of 23 patients with uSpA patients.In patients with PsA with DIP joint involvement there is increased frequency of HLA-B*15 (27.8%), HLA-B*44 (22.2%), HLA-B*57 (22.2%), HLA-C*6 (16.7%), HLA-C*7 (33.3%), and HLA-C*8 (33.3%). Whereas HLA-B*38 (43.8%), HLA-B*44 (18.8%), HLA-C*4 (43.7%) and HLA-C*7 (37.5%) were more frequent in undifferentiated spondyloarthritis. HLA-B*15 and HLA-B*57 both are detected in 2 out of 16 patients with uSpA.HLA-B*15 (27.3%) and HLA-B*38 (45.5%) were detected in an increased frequency in patients with uSpA who had FADES like skin lesions. In uSpA patients without FADES HLA-B*38 (41.7%) is detected in a lesser frequency. We speculate that HLA-B*38 may be unique to some ethnicities and there may be a distinct HLA-B*38 associated entity of uSpA.

We found that among 29 patients with psoriatic arthritis 2 (6.9%) were rheumatoid factor (RF) positive and 2 (6.9%) were anti-cyclic citrullinated peptide (anti-CCP) positive.

Among 23 patients with undifferentiated spondyloarthritis3 (13.0%) were RF positive and 1 (6.7%) was anti-CCP positive. One of three RF positive patients of uSpA group hadnail changes suggestive of psoriasis, FADES and DIP joints involvement. Another one had psoriatic like nail changes and the other one had family history of psoriasis, DIP joint involvement and psoriatic like nail changes. The patient who was anti-CCP positive, had marked DIP joint involvement and enthesitis.

Among the patients of uSpA who had RF and anti-CCP positive, two of them are HLA-B*57 positive, one is HLA-B*44 and other one was HLA-B*38 positive. Among the patients of psoriatic arthritis who were RF and Anti-CCP positive, one of them had HLA-B*13 and

HLA-B*51 positive and another one had HLAB*15 and HLA-B*40 positive.

Conclusion:
HLA-B*15, HLA-B*27, HLA-C*8 are alleles predominantly associated with psoriatic arthritis. A subset of patients of uSpA may carry a psoriatic arthritis genotype.

Limitations:
The study was conducted in a single center on small. Further research sample size needed to verify the results of our study.

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Conflicts of interest: Nothing to disclose.

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