








ORIGINAL ARTICLE

Influence of HLA-B27 on the Phenotype and Severity of Spondyloarthritis: A Study from a Tertiary Care Center in Nepal

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ABSTRACT

Introduction: Spondyloarthritis is a group of chronic inflammatory disorders characterized by shared clinical and genetic features, commonly involving the axial skeleton and strongly associated with HLA-B27. However, the clinical profile and disease severity of HLA-B27-negative axial spondyloarthritis remains poorly defined, particularly in South Asian populations. This study aimed to evaluate the association between HLA-B27 status and clinical phenotype, disease activity, functional status, inflammatory markers, and radiographic severity in Nepalese patients

Method: We conducted a cross-sectional study of adults with spondyloarthritis classified according to the ASAS 2009 criteria at a tertiary referral center in Kathmandu between October 16 2025 to April 15, 2026. Clinical features, laboratory parameters, disease activity indices, and radiographic findings were compared between HLA-B27-positive and HLA-B27-negative groups. Multivariate logistic regression was performed to identify independent associations.

Result: Out of 193 screened patients, 166 were included. The majority were male (80.3%), with a median age of 34 years. HLA-B27 was positive in 83.7% of patients. HLA-B27-negative patients presented at an older age (41 vs. 34 years) and had later symptom onset (32 vs. 25 years) compared to HLA-B27-positive patients. Family history was more common in HLA-B27-positive patients (32.4%, $p=0.06$). Dactylitis was significantly more frequent in HLA-B27-negative patients (33.3% vs. 8%, $p=0.001$) and remained independently associated (adjusted OR 6.25, 95% CI 1.9–20, $p=0.002$). No significant differences were observed in disease activity, inflammatory markers, or radiographic findings.

Conclusion: HLA-B27 positivity was associated with earlier disease onset, whereas HLA-B27-negative patients exhibited more peripheral features, particularly dactylitis, with similar overall disease severity. Further longitudinal studies are needed to validate these findings.

Key words: Axial spondyloarthritis; HLA B27; Clinical phenotype; Dactylitis

INTRODUCTION

Spondyloarthritis (SpA) comprises a group of interrelated chronic inflammatory rheumatic diseases, including ankylosing spondylitis, psoriatic arthritis, reactive arthritis, and arthritis associated with inflammatory bowel disease, which share overlapping clinical, radiologic, and genetic characteristics.^{1,2} Axial spondyloarthritis, defined by predominant involvement of the sacroiliac joints and spine, represents a major cause of chronic back pain and functional limitation in young and middle-aged adults, with substantial impact on quality of life and long-term disability.² A defining feature of many forms of axial SpA is the strong association with the class I major histocompatibility complex antigen HLA-B27, which has been implicated in both disease susceptibility and in shaping the clinical phenotype.^{3,4}

HLA B27 positivity is highly prevalent among patients with axial SpA, and its presence has historically been incorporated into classification criteria and used in clinical practice to support diagnosis in patients with suggestive symptoms.⁴ However, a significant minority of patients fulfil the criteria for axial SpA despite being HLA-B27 negative, and the extent to which these individuals exhibit distinct demographic characteristics, patterns of musculoskeletal involvement, or extra-articular manifestations remains incompletely understood.⁵ Existing data, largely derived from Western cohorts, suggest that HLA B27 status may influence age at onset, family history, and the distribution of axial versus peripheral disease, but findings have been

heterogeneous and may not be directly generalizable to other ethnic and genetic backgrounds. In South Asian populations, particularly in low-and middle-income countries, epidemiologic information on axial SpA and its genetic associations is limited, and the clinical profile of HLA-B27-negative disease is especially poorly characterized.^{5,6}

Against this background, we conducted a hospital-based cross-sectional study of adults fulfilling the Assessment of Spondyloarthritis International Society (ASAS) 2009 classification criteria for axial SpA at a tertiary referral center in Kathmandu, Nepal.⁴ The primary objective was to examine the relationship between HLA B27 status and clinical phenotype, including demographic characteristics, axial and peripheral musculoskeletal manifestations, extra-articular features, disease activity and functional indices, and radiographic sacroiliac joint involvement in this Nepalese cohort.

METHODS

Study design

We conducted a hospital-based cross-sectional observational study among adult patients (≥ 18 years) diagnosed with Spondyloarthritis according to ASAS classification criteria 2009⁷, attending the outpatient or inpatient department of Internal Medicine at Kathmandu Medical College, Sinamangal, Kathmandu, Nepal. After getting ethical approval from the Institutional Review Committee (IRC) [approval reference number: 15102025/04], we performed data collection among patients meeting our inclusion criteria presented

at our centre between October 16, 2025, and April 15, 2026.

Study participants

This study included adult patients (≥18 years) diagnosed with Spondyloarthritis (SpA) who presented to the Department of Internal Medicine at our centre.

Participants were eligible for inclusion if they: (i) fulfilled the Assessment of Spondyloarthritis International Society (ASAS) classification criteria 2009 for axial SpA; (ii) consented to undergo HLA-B27 testing as part of their clinical evaluation; and (iii) provided written informed consent for study participation and prospective follow-up assessments.

Patients were excluded for any of the following: (i) incomplete baseline clinical or laboratory data; (ii) a concurrent diagnosis of other systemic autoimmune or rheumatic diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus) that could confound the SpA phenotype; (iii) inability or unwillingness to provide informed consent; or (iii) the presence of significant comorbidities (e.g., advanced malignancy, active severe infection) that could independently influence disease severity assessment or clinical outcomes.

Variables and data curation

After informed consent from the patient, the data were obtained from medical records and prospective assessments using a structured case report form. HLA-B27 status was determined via polymerase chain reaction (PCR)-based genotyping⁸, performed in accordance with standard laboratory protocols. In each case report, clinical data including gender, current age, age at disease onset, duration of symptoms, and patient symptoms like lower back pain, peripheral arthritis (joint involvement pattern), enthesitis (tendon or ligament inflammation), extra-articular manifestations (uveitis, psoriasis, inflammatory bowel disease) were collected. Similarly, information on laboratory parameters like CRP and ESR, and questionnaire-based assessment parameters for disease severity, such as BASDAI (Bath Ankylosing Spondylitis Disease Activity Index), BASMI, and ASDAS-CRP, were collected. Radiographic findings were evaluated based on sacroiliac joint imaging using the modified New York criteria.⁹ Modified Schober and chest expansion were also measured. All data were anonymised upon collection and stored securely in a password-protected database for subsequent analysis.

Sampling method and Sample size:

The sampling method was purposive sampling. The minimum sample size was calculated using the single population proportion formula $n = [Z^2pq]/d^2$, assuming a 95% confidence level ($Z = 1.96$), assumed prevalence of HLA-B27 positivity in axial SpA of 70%, a precision of 10%, yielding a calculated sample size of 81 participants.

Data analysis

The data were entered into a Microsoft Excel sheet in encoded form and later exported to Epi Info version 7.2.4 and licensed STATA 15 software for further analysis. Categorical data were presented using appropriate charts and tables, with frequencies and percentages. Normally distributed quantitative data were reported as the mean with a standard deviation. Non-normally distributed continuous data were reported as median (interquartile range). Chi-square test, Fisher's exact test, and Mann-Whitney U test were used to compare two groups (HLA-B27- positive and negative). The level of significance was set at 5%, and a p-value < 0.05 was considered statistically significant.

RESULTS

Cohort Characteristics and Demographics

Over a 6-month period, a total of 193 patients with axial spondyloarthritis presented to our centre; among them, 166 patients meeting our eligibility criteria were included in our study. The flow diagram of our study is depicted in Figure 1. The overall population was male-dominant, with 134 male patients (80.7%) and 32 female patients (19.3%). The median age of our study population was 34 years (range 28-42), and the average age at symptom onset was 25 years (range 20-32). All the patients were below the age of 45 years at the time of onset of symptoms, and all had chronic inflammatory lower back pain at presentation. The human leukocyte antigen B27 (HLA-B27) was positive in most patients: 139 (83.7%) tested positive, and 27 (16.3%) tested negative. Hypertension was identified as the most common comorbidity, followed by diabetes mellitus. Descriptive statistics for the entire cohort are summarized in Table 1. The cohort exhibited moderate disease activity, as reflected by standard indices: a Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) of 2.5 (1.3-4.0), an Ankylosing Spondylitis Disease Activity Score using C-reactive protein (ASDAS-CRP) of 2.6 (1.8-3.6), and a Bath Ankylosing Spondylitis Metrology Index (BASMI) of 1.65 (0.6-3.25).

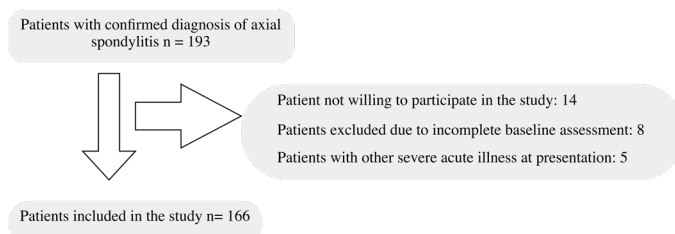


Figure 1: Flowchart showing inclusion and exclusion of patients in our cohort

Table 1: Biological characteristics of our study population (n=166)

Characteristics	n	
Age at presentation (years)	34	[28-42]
Age at onset of symptoms (years)	25.5	[20-32]
Duration of illness (months)	78	[48-121]
Gender		
Male	134	80.7%
Female	32	19.3%
Comorbidities		
DM	16	9.6%
HTN	31	18.7%
CAD	2	1.2%
Personal history		
Smoking	26	15.7%
Alcohol	25	15.1%
Family history		
Positive	49	29.5%
HLA B27		
Positive	139	83.7%
Negative	27	16.3%
Other lab parameters		
ESR	24	[12-43]
CRP	12	[5-29.2]

*DM, Diabetes mellitus; HTN, Hypertension; CAD, Coronary artery disease; ESR, Erythrocyte sedimentation rate; CRP, C-reactive protein

Comparative Analysis of Clinical Features by HLA-B27 Status

The patients were stratified into HLA-B27-positive and HLA-B27-negative groups. The associations between HLA-B27 status and a range of categorical clinical features, including demographics, clinical history, and specific disease manifestations, were evaluated. HLA-B27-negative patients presented at an older median age [41 vs. 34 years] and reported a later median age of disease onset [32 vs. 25 years] compared to HLA-B27-positive patients. Similarly, a strong inverse relationship was observed between HLA-B27 positivity and the presence of peripheral inflammatory features. Dactylitis was present in over a quarter of HLA-B27-negative patients (9/27, 33.3%) but was significantly less common in the HLA-B27-positive group (11/139, 8%; p=0.010). These findings suggest that HLA-B27-negative status may be associated with a higher propensity for these specific peripheral manifestations. A significant association was also found between HLA-B27 status and a diagnosis of inflammatory bowel disease (IBD)-associated SpA ($\chi^2 = 9.853, p = 0.020$). In multivariate logistic regression (Table 2) adjusting for age, gender, smoking status, disease duration, presence of diabetes and hypertension, HLA-B27 positivity remained independently associated with significantly lower odds of dactylitis (adjusted OR = 0.16, 95% CI: 0.05-0.52, p=0.002). None of the other covariates reached statistical significance.

Table 2: Multivariate logistic regression analysis of factors associated with dactylitis in patients with axial spondyloarthritis

Predictor	Adjusted OR	95% CI	p-value
HLA-B27 positive	0.16	0.05-0.52	0.002*
Age	1.02	0.98-1.06	0.34
Male sex	1.18	0.42-3.29	0.76
Smoking	1.41	0.53-3.78	0.49
Disease duration	0.99	0.97-1.01	0.31
Diabetes mellitus	0.88	0.24-3.22	0.85
Hypertension	1.09	0.39-3.01	0.87

OR: odds ratio, CI: confidence interval

No statistically significant differences were found between HLA-B27-positive and HLA-B27-negative patients for other clinical features. Although a positive family history of spondyloarthritis was more frequently observed in HLA-B27-positive patients (32.4% vs. 14.8%), this difference did not reach statistical significance. The presence of axial and enthesal features, such as enthesitis, costochondritis, and plantar fasciitis, other peripheral features, like synovitis, and extra-articular manifestations, such as uveitis, psoriasis, and scleritis, didn't show a significant difference with HLA B27 status in our study population. No gender predilection difference seen with HLA-B27 status (p=0.696). The detailed results for these variables are presented in Table 3. Measures of current disease activity, inflammation, and function, including BASDAI, ASDAS scores, BASMI, ESR, and CRP, showed minimal descriptive differences between the two groups as depicted on Table 3.

Feature	HLA-B27-negative (n=27)	HLA-B27-positive (n=139)	p value
Gender (Male)	23 (85.2%)	111 (79.9%)	0.500
Female	4 (14.8%)	28 (20.1%)	
Age (years)	41 (31-41)	34 (27-40)	0.040*
Age at Onset (years)	32 (21-38)	25 (20-30)	0.007*

Feature	HLA-B27-negative (n=27)	HLA-B27-positive (n=139)	p value
Duration of illness (months)	71 (36-114)	89 (48-121)	0.740
Family history			
Positive family history	4 (14.8%)	45 (32.4%)	0.060
Clinical diseases at presentation			
Enthesitis	19 (70.4%)	86 (61.9%)	0.400
Dactylitis	9 (33.3%)	11 (8%)	0.001*
Psoriasis	2 (10.5%)	10 (7.5%)	0.640
Plantar fasciitis	6 (22.2%)	39 (28%)	0.700
Peripheral synovitis	12 (44.4%)	61 (43.9%)	0.950
Costochondritis	6 (22.2%)	28 (20.1%)	0.800
Uveitis	3 (11.1%)	31 (22.3%)	0.220
Inflammatory Bowel Disease	2 (7.4%)	3(2.2%)	0.300
Radiographic disease on X ray pelvis			
Radiographic disease	20 (74.1%)	104 (74.8%)	0.900
Phenotype			
Ankylosing Spondylitis	17 (63%)	94 (67.6%)	-
Psoriatic Arthritis	3 (11.1%)	10 (7.2%)	0.630
Enteropathic Arthritis	2 (7.4%)	3 (2.2%)	0.120
Undifferentiated SpA	5 (18.5%)	32 (23.0%)	0.110
Disease severity parameters			
ESR	21 (10- 42)	24 (13-43)	0.550
CRP	12 (8-28)	12 (5-30)	0.820
BASDAI score	2.3 (1.6-3.2)	2.7 (1.2-4.2)	0.510
BASMI score	1.4 (0.4-3)	1.8 (0.6-3.4)	0.310
ASDAS-CRP	2.1 (1.8-3.0)	2.7 (1.8-3.6)	0.220

* Denotes statistical Significance (p<0.05). * SpA: Spondyloarthritis; BASDAI: Bath ankylosing Spondylitis disease activity index; BASMI: Bath ankylosing Spondylitis Metrology index; ASDAS-CRP, Ankylosing Spondylitis disease activity score using C-reactive Protein

DISCUSSION

In this study, we analysed the clinical characteristics of axial SpA patients according to HLA-B27 status. Among 166 patients included, males accounted for 80.3% of the study cohort. Similar results were also described in the REGISPONDER, SPARCC, and PSOAS registries.^{10,11} Male predominance in axial spondyloarthritis may be due to a combination of higher penetrance of HLA-B27 in males, pro-inflammatory hormonal influences, and greater radiographic progression, facilitating earlier recognition.¹² Our study showed that the prevalence of HLA-B27 was 83.7%. A systematic review found that among axSpA patients fulfilling any of the contemporary criteria (New York, ESSG, or ASAS), the prevalence of HLA-B27 varies between 26.2% and 91%.¹³ The higher prevalence of HLA-B27-positive cases in axial spondyloarthritis is due to a strong genetic association with disease susceptibility and its inclusion in classification criteria, which also contributes to diagnosis bias.⁴

We observe that the mean age of onset of axial SpA was 9 years earlier in patients with HLA- B27 than in those HLA-B27-negative (24 vs 33). This may be attributed to a stronger genetic predisposition and earlier activation of inflammatory pathways, particularly the IL-17/IL-23 axis. In contrast, HLA-B27-negative patients often have a more heterogeneous pathogenesis with weaker genetic influence,

leading to a later onset.¹² Our findings indicate that HLA-B27-positive patients had an earlier median age of onset compared to HLA-B27-negative patients (32 vs 41 years). Additionally, our results showed that HLA-B27-positive SpA patients had greater family aggregation than HLA-B27-negative patients, which is consistent with previous studies in radiographic axial SpA and in early forms of SpA patients from DESIR.^{14,15} In addition, the presence of a family history in 14.8% of HLA-B27-negative patients is consistent with earlier research, indicating that non-HLA-B27 genetic contributors may play a role in disease aggregation.^{14,15}

There was some difference in musculoskeletal manifestations such as costochondritis, enthesitis, and plantar fasciitis between the groups; however, dactylitis was significantly more common in HLA-B27-negative patients compared to HLA-B27-positive patients (33.3% vs 8%). The higher prevalence of dactylitis in HLA-B27-negative patients may be due to a greater tendency toward peripheral and enthesitis-related inflammation rather than axial disease, reflecting a different immunopathological phenotype.¹⁶ A recent study done in Spain showed a higher frequency of dactylitis and extraarticular manifestations in accordance with ours.¹⁶ Our study showed the prevalence of uveitis is higher in HLA-B27-positive patients than in HLA-B27-negative patients, which is in accordance with a previous study.¹⁷ In contrast, another previous study by Correia et al. showed no significant difference.¹⁸

We observe that the prevalence of psoriasis and IBD was lower in HLA-B27-positive patients than in HLA-B27-negative patients, which was consistent with previous studies.^{10,14} However, no clear evidence has been found to prove that the risk of developing IBD is higher in axial SpA patients with HLA-B27.¹⁹ The negative association of psoriasis with HLA-B27 may be due to selection bias, as patients HLA-B27-negative need more extra-articular features to meet Amor criteria for spondyloarthritis.¹⁰

Regarding disease severity, our findings showed no significant differences. While some studies have reported comparable BASDAI and BASFI scores between HLA-B27-negative and positive patients^{20,21}, others have demonstrated worse scores in HLA-B27-negative individuals.¹⁰ In our study, ESR, CRP, BASDAI, BASFI, and ASDAS-CRP scores were slightly higher in HLA-B27-positive patients compared to those HLA-B27-negative, which is consistent with the study done by Arévalo et al.¹⁰ However, a positive correlation between the presence of HLA-B27 and high ASDAS-CRP was not previously confirmed.^{10,22} We observe that patients who are HLA-B27 positive have a higher frequency of earlier disease onset, lower median age, less dactylitis, and a greater prevalence of family history compared to HLA-B27-negative patients.

The study's small HLA-B27-negative subgroup (n=27), combined with its single-centre, cross-sectional design, limits statistical power, prevents causal and longitudinal inferences, introduces potential unmeasured confounding factors (such as treatment effects and socioeconomic variables), and restricts the generalizability of findings to the Nepalese hospital population.

CONCLUSION

This study highlights distinct clinical phenotypes associated with HLA-B27 status in Nepalese patients with axial spondyloarthritis. While HLA-B27 positivity predominates and correlates with earlier disease onset, HLA-B27 negativity appears associated with a higher burden of peripheral inflammatory features, particularly dactylitis. Future longitudinal, multicentre studies with larger sample

sizes are needed to confirm these associations, explore their underlying mechanisms, and evaluate their prognostic significance.

REFERENCES

1. Taitt HA, Balakrishnan R. Spondyloarthritis. *Immunology and Allergy Clinics of North America*. 2023;43(3):593–612. Available from: <https://doi.org/10.1016/j.iac.2022.10.001>
2. Sieper J, Poddubnyy D. Axial spondyloarthritis. *The Lancet*. 2017;390(10089):73–84. Available from: [https://doi.org/10.1016/s0140-6736\(16\)31591-4](https://doi.org/10.1016/s0140-6736(16)31591-4).
3. Taurog JD. Arthritis in HLA-B27 transgenic animals. *The American Journal of the Medical Sciences*. 1998;316(4):250–6. Available from: <https://doi.org/10.1097/0000441-199810000-00005>.
4. Rudwaleit M, Van Der Heijde D, Landewé R, Listing J, Akkoc N, Brandt J, Braun J, et al. The development of Assessment of SpondyloArthritis international Society classification criteria for axial spondyloarthritis (part II): validation and final selection. *Annals of the Rheumatic Diseases*. 2009;68(6):777–83. Available from: <https://doi.org/10.1136/ard.2009.108233>.
5. Rudwaleit M, Haibel H, Baraliakos X, Listing J, Märker-Hermann E, Zeidler H, et al. The early disease stage in axial spondylarthritis: Results from the german spondyloarthritis inception cohort. *Arthritis & Rheumatism*. 2009;60(3):717–27. Available from: <https://doi.org/10.1002/art.24483>.
6. Malaviya AN. Spondyloarthritis in India. *Indian Journal of Rheumatology*. 2020;15(1S). Available from: <https://doi.org/10.4103/0973-3698.284742>.
7. Sieper J, Rudwaleit M, Baraliakos X, Brandt J, Braun J, Burgos-Vargas R, et al. The Assessment of SpondyloArthritis international Society (ASAS) handbook: a guide to assess spondyloarthritis. *Annals of the Rheumatic Diseases*. 2009 May 11;68:ii1–44. Available from: <https://doi.org/10.1136/ard.2008.104018>.
8. Skalska U, Kozakiewicz A, Maśliński W, Jurkowska M. HLA-B27 detection - comparison of genetic sequence-based method and flow cytometry assay. *Reumatologia* 2015; 53(2):74-8. PMID: 27407231.
9. Gaillard F, Hng J, Kaya H. Sacroiliitis grading (New York criteria). *Radiopaedia.org*. 2008 May 2; Available from: <https://doi.org/10.53347/rid-2013>.
10. Group R, Arévalo M, Masmitjà JG, Moreno M, Calvet J, Orellana C, et al. Influence of HLA-B27 on the Ankylosing Spondylitis phenotype: results from the REGISPONSER database. *Arthritis Research & Therapy*. 2018;20(1):221. Available from: <https://doi.org/10.1186/s13075-018-1724-7>.
11. Maksymowych WP. Imaging in axial spondyloarthritis. *Rheumatic Disease Clinics of North America*. 2016;42(4):645–62. Available from: <https://doi.org/10.1016/j.rdc.2016.07.003>.
12. Reveille JD. The genetic basis of spondyloarthritis. *Annals of the Rheumatic Diseases*. 2011;70:i44–50. Available from: <https://doi.org/10.1136/ard.2010.140574>.
13. Ziade NR. HLA B27 antigen in Middle Eastern and Arab countries: systematic review of the strength of association with axial spondyloarthritis and methodological gaps. *BMC Musculoskeletal Disorders*. 2017;18(1):280. Available from: <https://doi.org/10.1186/s12891-017-1639-5>
14. Tsui F, Tsui HW, Akram A, Haroon N, Inman R. The genetic basis of ankylosing spondylitis: new insights into disease pathogenesis. *The Application of Clinical Genetics*. 2014;7:105. Available from: <https://doi.org/10.2147/tacg.s37325>.
15. Chung HY, Machado P, Van Der Heijde D, D'Agostino

- MA, Dougados M. HLA-B27 positive patients differ from HLA-B27 negative patients in clinical presentation and imaging: results from the DESIR cohort of patients with recent onset axial spondyloarthritis. *Annals of the Rheumatic Diseases*. 2011;70(11):1930–6. Available from: <https://doi.org/10.1136/ard.2011.152975>.
16. Yang M, Xu M, Pan X, Hu Z, Li Q, Wei Y, et al. Epidemiological comparison of clinical manifestations according to HLA-B*27 carrier status of Chinese Ankylosing Spondylitis patients. *Tissue Antigens*. 2013;82(5):338–43. Available from: <https://doi.org/10.1111/tan.12186>.
 17. Accorinti M, Iannetti L, Liverani M, Caggiano C, Gilardi M. Clinical features and prognosis of HLA B27-associated acute anterior uveitis in an Italian patient population. *Ocular Immunology and Inflammation*. 2010;18(2):91–6. Available from: <https://doi.org/10.3109/09273941003597268>.
 18. Correia MSR, Portela M, Baptista M, Cordeiro M, Guedes M. Clinical features and outcomes of HLA-B27-positive and HLA-B27-negative acute anterior uveitis in a tertiary care center. *The Pan-American Journal of Ophthalmology*. 2025;7(1). Available from: https://doi.org/10.4103/pajo.pajo_18_25.
 19. Fahed H, Mauro D, Ciccio F, Ziade NR. What Does Human Leukocyte Antigen B27 Have to Do with Spondyloarthritis? *Rheumatic Disease Clinics of North America*. 2020;46(2):225–39. Available from: <https://doi.org/10.1016/j.rdc.2020.01.002>.
 20. Kim TJ, Kim TJ, Kim TH, Kim TH. Clinical spectrum of ankylosing spondylitis in Korea. *Joint Bone Spine*. 2010;77(3):235–40. Available from: <https://doi.org/10.1016/j.jbspin.2009.11.015>.
 21. Jaakkola E, Herzberg I, Laiho K, Barnardo MCNM, Pointon JJ, Kauppi M, et al. Finnish HLA studies confirm the increased risk conferred by HLA-B27 homozygosity in ankylosing spondylitis. *Annals of the Rheumatic Diseases*. 2005;65(6):775–80. Available from: <https://doi.org/10.1136/ard.2005.041103>.
 22. Sheehan NJ. HLA-B27: what's new? *Rheumatology* 2010; 49(4):621–631. doi: 10.1093/rheumatology/kep450. PMID: 20100794.

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AUTHOR CONTRIBUTIONS

Prabin Khatri took the overall responsibility for the study, including conceptualization, methodology development, analysis, and finalization of the manuscript. All other authors contributed equally in data collection, statistical analysis, and manuscript preparation.

CONFLICT OF INTEREST

The authors declare no competing interests

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