

Original Research Article

Rural–urban differences in disease activity and vitamin D status in ankylosing spondylitis

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ABSTRACT

Background: Vitamin D has important immunomodulatory effects and may influence disease activity in ankylosing spondylitis (AS). Rural–urban differences in sunlight exposure, diet, and healthcare access may affect vitamin D status. This study evaluated the impact of residential background on serum vitamin D levels and disease activity among patients with AS in Northeast India.

Methods: This cross-sectional observational study included 40 consecutive patients fulfilling the ASAS 2009 classification criteria for AS at Assam Medical College and Hospital, Dibrugarh (2024–2025). Serum 25-hydroxyvitamin D [25(OH)D] was measured by enzyme-linked fluorescent assay (ELFA). Disease activity was assessed using BASDAI, ASDAS-ESR, and ASDAS-CRP. Rural and urban groups were compared using the Mann–Whitney U test, and correlations were assessed using Spearman's rank correlation.

Results: The mean age was 41.1 ± 14.1 years, with 55% males; 62.5% were from rural areas. Vitamin D deficiency, insufficiency, and normal levels were observed in 45%, 32.5%, and 22.5% of patients, respectively. Mean serum 25(OH)D levels were similar in rural and urban groups (15.3 ± 5.8 vs. 14.7 ± 6.1 ng/mL; $p=0.715$). High disease activity (BASDAI ≥ 4) was present in 76.0% and 80.0% of rural and urban patients, respectively, with no significant differences in BASDAI or ASDAS-ESR scores. Serum vitamin D levels showed significant negative correlations with BASDAI, ASDAS-ESR, ASDAS-CRP, ESR, and CRP (all $p < 0.001$).

Conclusions: Vitamin D deficiency was common and strongly associated with higher disease activity, although no significant rural–urban differences were observed. Routine assessment of vitamin D may be beneficial in patients with AS irrespective of residential background.

Keywords: Ankylosing spondylitis, Vitamin D, BASDAI, ASDAS, Rural–urban, Northeast India

INTRODUCTION

Ankylosing spondylitis (AS) represents the classical form of axial spondyloarthritis, a chronic immune-mediated inflammatory disorder that primarily affects the sacroiliac joints and vertebral column. The disease commonly manifests with inflammatory low back pain, gradually progressive spinal rigidity, and eventual ankylosis. AS usually begins during early adulthood, particularly in the second and third decades of life, and is associated with

considerable functional impairment, diminished quality of life, and significant socioeconomic burden.^{1,2} In the Indian population, the estimated prevalence of AS is around 1.5 per 1000 individuals, occurring more frequently in males and showing a strong association with HLA-B27 positivity.³

The underlying pathogenesis of AS is driven by immune dysregulation involving the IL-23/IL-17 pathway, inflammation at enthesal sites, and abnormal

osteoproliferation. These mechanisms are influenced by interactions among genetic susceptibility, environmental exposures, and microbial factors.⁴ Vitamin D has emerged as an environmental factor of growing interest because of its dual role in skeletal metabolism and immune modulation. As a fat-soluble secosteroid hormone, vitamin D exerts immunological effects through vitamin D receptors (VDRs), which are widely distributed across immune cell populations. The active metabolite, 1,25-dihydroxyvitamin D, suppresses Th17 cell differentiation, reduces IL-17A and IL-23 production, and enhances regulatory T-cell (Treg) activity, all of which are relevant to the inflammatory pathways implicated in AS.^{5,6}

Despite plentiful sunlight, vitamin D deficiency remains highly prevalent across South and Southeast Asia. Contributing factors include inadequate dietary intake, reduced outdoor exposure, darker skin pigmentation, and environmental pollution.⁷ However, relatively little attention has been given to the influence of residential setting particularly rural versus urban habitation on vitamin D status and disease severity among patients with AS. Individuals residing in rural areas of Assam, many of whom engage in agricultural or outdoor occupations, may theoretically have greater sunlight exposure and consequently higher vitamin D levels compared to urban residents who spend more time indoors.

In contrast, urban populations may benefit from improved healthcare accessibility and more diverse nutritional intake. The interaction of these opposing influences makes rural–urban comparisons clinically relevant and biologically intriguing.

Northeast India, especially Assam, offers a unique setting for such research due to its high solar exposure, marked ethnic diversity, predominantly rural population distribution, and limited specialist rheumatology services. Assam Medical College and Hospital (AMCH), Dibrugarh, functions as a major tertiary referral center for Upper Assam and caters to patients from both urban Dibrugarh and surrounding remote rural districts. Against this background, the present study was undertaken to evaluate differences in serum 25-hydroxyvitamin D [25(OH)D] concentrations and validated disease activity scores between rural and urban AS patients attending AMCH, while also examining the overall association between vitamin D status and disease activity in the study population.

METHODS

Study design and setting

A hospital-based cross-sectional observational study was carried out in the Department of Medicine at Assam Medical College and Hospital, Dibrugarh, Assam, between 2024 and 2025.

Participants

The study included 40 consecutive patients diagnosed with ankylosing spondylitis according to the Assessment of Spondyloarthritis International Society (ASAS) 2009 classification criteria.⁹ Participants were recruited purposively from the rheumatology and general medicine outpatient departments of AMCH. Only adults aged 18 years or older were eligible for inclusion. Exclusion criteria comprised: Established malabsorption disorders; chronic kidney disease with estimated glomerular filtration rate (eGFR) below 45 mL/min/1.73 m²; Active liver disease; Recent use of vitamin D supplements, glucocorticoids, or antiepileptic medications within the preceding three months; and Pregnancy or lactation.

Data collection

Information was recorded using a structured case record form that captured sociodemographic characteristics including age, sex, residential background, and socioeconomic status assessed using the Modified Kuppuswamy Scale. Additional variables such as smoking history, coexisting illnesses, family history of AS or autoimmune disease, and duration of illness were also documented. Residential status was categorized as rural or urban according to the patient's registered address and official census classification, with villages and block-level areas considered rural, and towns or cities classified as urban.

Laboratory investigations

Fasting venous blood samples were obtained during morning hours between 08:00 and 10:00 AM. Serum 25-hydroxyvitamin D [25(OH)D] levels were estimated using enzyme-linked fluorescent assay (ELFA) on the VIDAS® platform (bioMérieux), which demonstrated intra- and inter-assay variability below 8%. Vitamin D levels were interpreted according to Endocrine Society criteria as deficiency (<20 ng/ml), insufficiency (20–29 ng/ml), and sufficiency (≥30 ng/ml).¹⁰ Serum C-reactive protein (CRP) was measured using high-sensitivity immunoturbidimetric methods, while erythrocyte sedimentation rate (ESR) was determined using the Westergren technique.

Disease activity assessment

Disease activity was evaluated using two validated scoring systems.

Bath ankylosing spondylitis disease activity index (BASDAI)

BASDAI is a patient-reported outcome measure encompassing six domains: fatigue, spinal pain, peripheral joint pain, enthesitis, and the severity and duration of morning stiffness. Scores range from 0 to 10, with values ≥4 indicating active disease.

Ankylosing spondylitis disease activity score (ASDAS)

ASDAS was calculated using both ESR-based (ASDAS-ESR) and CRP-based (ASDAS-CRP) formulas. These incorporate patient-reported measures such as spinal pain, peripheral pain/swelling, and global assessment together with inflammatory markers. Disease activity categories were defined as: inactive (<1.3), moderate (1.3–<2.1), high (2.1–<3.5), and very high (≥ 3.5).

Statistical analysis

Data were entered in Microsoft Excel and analysed using SPSS version 29.0 (IBM Corp.). Continuous variables are reported as mean $\pm$ standard deviation (SD). The Shapiro–Wilk test assessed normality. Since most continuous variables were not normally distributed, the Mann–Whitney U test was used for rural–urban comparisons. Categorical variables were compared with Pearson's chi-square test or Fisher's exact test as appropriate. Spearman's rank correlation (r_s) was used to evaluate the association

between serum 25(OH)D and disease activity indices. A two-tailed $p < 0.05$ was considered statistically significant.

Ethical clearance

Ethical approval was obtained from the Institutional Ethics Committee, AMCH (Human). Written informed consent was obtained from all participants.

RESULTS*Baseline characteristics*

Forty patients with AS were enrolled: 25 (62.5%) from rural areas and 15 (37.5%) from urban areas. The mean age of the cohort was 41.1 ± 14.1 years (range: 20–70 years). Males constituted 55% of participants overall; rural patients were more often male (60.0% vs. 46.7%). The mean disease duration was 10.9 ± 5.7 years. Baseline characteristics stratified by area of residence are presented in Table 1.

Table 1: Baseline sociodemographic and clinical characteristics by area of residence (n=40).

Variables	Rural (n=25)	Urban (n=15)	Total (n=40)	P value
Age (years), mean $\pm$ SD	39.7 $\pm$ 14.8	43.5 $\pm$ 13.7	41.1 $\pm$ 14.1	0.421
Male gender, N (%)	15 (60.0%)	7 (46.7%)	22 (55.0%)	0.399
Female gender, N (%)	10 (40%)	8 (53.3%)	18 (45%)	
Socioeconomic status, N (%)				0.312
Lower and lower middle	13 (52.0%)	6 (40.0%)	19 (47.5%)	
Middle and upper middle	8 (32.0%)	5 (33.3%)	13 (32.5%)	
Upper	4 (16.0%)	4 (26.7%)	8 (20.0%)	
Disease duration (years), mean $\pm$ SD	10.5 $\pm$ 5.8	11.6 $\pm$ 5.7	10.9 $\pm$ 5.7	0.538
Smoker, N (%)	11 (44.0%)	6 (40.0%)	17 (42.5%)	0.800
Family history of AS/autoimmune disease, N (%)	8 (32.0%)	2 (13.3%)	10 (25.0%)	0.178
Co-morbidities, N (%)	11 (44.0%)	7 (46.7%)	18 (45.0%)	0.863
Hypertension	5 (20.0%)	4 (26.7%)	9 (22.5%)	
Diabetes mellitus	1 (4.0%)	2 (13.3%)	3 (7.5%)	
Hypothyroidism	1 (4.0%)	1 (6.7%)	2 (5.0%)	

SD = standard deviation. p values: Mann–Whitney U test for continuous variables; chi-square or Fisher's exact test for categorical variables.

Table 2: Vitamin D status by area of residence (n=40).

Vitamin D category	Cut-off (ng/ml)	Rural (n=25)	Urban (n=15)	Total (n=40)	P value
Deficiency	<20	10 (40.0%)	8 (53.3%)	18 (45.0%)	
Insufficiency	20–29	10 (40.0%)	3 (20.0%)	13 (32.5%)	0.425
Mean 25(OH)D, mean $\pm$ SD	—	15.3 $\pm$ 5.8	14.7 $\pm$ 6.1	15.1 $\pm$ 5.9	0.715

25(OH)D = serum 25-hydroxyvitamin D. Categorical comparison: chi-square test. Continuous comparison: Mann–Whitney U test.

Vitamin D status

Overall, the mean serum 25(OH)D was 15.1 ± 5.9 ng/ml. Vitamin D deficiency (<20 ng/ml) was present in 18/40 patients (45.0%), insufficiency (20–29 ng/ml) in 13/40 (32.5%), and normal levels (≥ 30 ng/ml) in only 9/40 (22.5%). Among rural patients, 40.0% (n=10) were

deficient and 40.0% (n=10) insufficient. In the urban group, deficiency was more frequent (53.3%), while insufficiency was less common (20.0%). However, this distribution did not reach statistical significance ($\chi^2=1.71$, $df=2$, $p=0.425$). Mean serum 25(OH)D was comparable between groups (Rural: 15.3 ± 5.8 ng/ml; Urban: 14.7 ± 6.1 ng/ml; $p=0.715$). Results are summarized in Table 2.

Disease activity indices

The majority of patients had active disease. BASDAI-defined active disease (≥ 4) was found in 31/40 patients (77.5%) overall 76.0% of rural and 80.0% of urban patients ($p=1.000$). The mean BASDAI score was

5.13 ± 1.24 in rural patients and 5.31 ± 1.30 in urban patients ($p=0.664$). Similarly, ASDAS-ESR and ASDAS-CRP scores were comparable between groups. Inflammatory markers (ESR and CRP) were also similar. Detailed comparisons are given in Table 3, 4 and 5.

Table 3: Comparison of disease activity indices and inflammatory markers by area of residence (n=40).

Parameters	Rural (n=25), Mean $\pm$ SD	Urban (n=15), Mean $\pm$ SD	P value
BASDAI score	5.13 $\pm$ 1.24	5.31 $\pm$ 1.30	0.664
Active disease (BASDAI ≥ 4), N (%)	19/25 (76.0%)	12/15 (80.0%)	1.000
ASDAS-ESR score	3.38 $\pm$ 1.89	3.07 $\pm$ 1.95	0.327
ASDAS-CRP score	3.37 $\pm$ 1.80	3.18 $\pm$ 2.24	0.484
ESR (mm/h)	30.9 $\pm$ 12.9	33.5 $\pm$ 15.5	0.867

BASDAI = Bath Ankylosing Spondylitis Disease Activity Index; ASDAS = AS Disease Activity Score; ESR = erythrocyte sedimentation rate; CRP = C-reactive protein. Mann–Whitney U test for all comparisons.

Table 4: Distribution of ASDAS-ESR disease activity categories by area of residence (n=40).

ASDAS-ESR category	Rural (n=25)	Urban (n=15)	Total (n=40)
Inactive (<1.3)	2 (8.0%)	1 (6.7%)	3 (7.5%)
Moderate (1.3–<2.1)	8 (32.0%)	6 (40.0%)	14 (35.0%)
High (2.1–<3.5)	5 (20.0%)	2 (13.3%)	7 (17.5%)
Very high (≥ 3.5)	10 (40.0%)	6 (40.0%)	16 (40.0%)

$p = 0.892$ (Fisher's exact test). ASDAS = Ankylosing Spondylitis Disease Activity Score.

Table 5 Distribution of ASDAS-ESR disease activity categories by area of residence (n=40).

ASDAS-CRP category	Rural (n=25)	Urban (n=15)	Total (n=40)
Inactive (<1.3)	2 (8.0%)	3 (20.0%)	5 (12.5%)
Moderate (1.3–<2.1)	8 (32.0%)	4 (26.7%)	12 (30.0%)
High (2.1–<3.5)	5 (20.0%)	3 (20%)	8 (20.0%)
Very high (≥ 3.5)	10 (40.0%)	5 (33.3%)	15 (37.5%)

$p = 0.734$ (Fisher's exact test). ASDAS = Ankylosing Spondylitis Disease Activity Score.

Table 6: Spearman's rank correlation of serum 25(OH)D with disease activity indices (n=40).

Disease activity parameter	Spearman's r_s	95% CI	P value	Strength
BASDAI score	−0.814	−0.893 to −0.695	<0.001	Strong inverse
ASDAS-ESR score	−0.715	−0.837 to −0.548	<0.001	Strong inverse
ASDAS-CRP score	−0.615	−0.769 to −0.407	<0.001	Moderate-strong inverse
ESR (mm/h)	−0.765	−0.871 to −0.614	<0.001	Strong inverse
CRP (mg/l)	−0.872	−0.932 to −0.776	<0.001	Very strong inverse

25(OH)D = serum 25-hydroxyvitamin D. Negative r_s indicates: higher vitamin D $\rightarrow$ lower disease activity. All $p < 0.001$.

Table 7: Mean disease activity scores by vitamin D status category full cohort (n=40).

Parameter	Deficiency (n=18)	Insufficiency (n=13)	Normal (n=9)	P value
Mean 25(OH)D (ng/ml)	10.1 $\pm$ 2.3	17.2 $\pm$ 1.9	24.1 $\pm$ 1.5	<0.001
Mean BASDAI	6.3 $\pm$ 0.8	4.7 $\pm$ 0.7	3.7 $\pm$ 0.3	<0.001
Mean ASDAS-ESR	4.7 $\pm$ 1.6	2.3 $\pm$ 0.8	1.8 $\pm$ 0.3	<0.001
Mean ASDAS-CRP	4.6 $\pm$ 1.9	2.5 $\pm$ 0.9	2.0 $\pm$ 0.7	<0.001
Mean CRP (mg/l)	16.3 $\pm$ 6.1	7.0 $\pm$ 2.5	3.0 $\pm$ 0.7	<0.001
Mean ESR (mm/h)	41.2 $\pm$ 12.6	25.5 $\pm$ 6.4	17.8 $\pm$ 1.8	<0.001
Active disease, N (%)	18/18 (100%)	10/13 (76.9%)	3/9 (33.3%)	<0.001

Values in red indicate statistically significant findings. Kruskal–Wallis test for continuous variables; chi-square for categorical. Post-hoc Dunn's test confirmed pairwise significance ($p < 0.05$ for all pairs).

DISCUSSION

This cross-sectional study, conducted at the only tertiary care centre serving a large rural–urban population in Upper Assam, investigated whether area of residence a rarely examined variable in AS research meaningfully modifies vitamin D status or disease activity. Our principal finding is that rural and urban AS patients showed no statistically significant difference in either serum 25(OH)D levels or disease activity indices, yet vitamin D deficiency was highly prevalent and strongly inversely correlated with disease burden across the entire cohort.

Prevalence of vitamin D deficiency

The overall vitamin D deficiency prevalence of 45% (n=18) in our AS cohort is consistent with published Indian data. Aggarwal et al reported deficiency in 39% of AS patients at a North Indian centre, while a meta-analysis by Zhao et al demonstrated that AS patients have significantly lower 25(OH)D levels than healthy controls (WMD −5.9 ng/ml, $p<0.001$).^{11,12} The elevated prevalence of deficiency in our cohort despite the sun-rich northeast Indian climate likely reflects the confluence of high skin pigmentation limiting UV-B cutaneous synthesis, rural patients' increased coverage of skin during agricultural activities, and urban patients' predominantly indoor lifestyles with reduced sun exposure. These competing mechanisms may explain why our rural–urban comparison yielded no significant difference both groups faced distinct but balancing barriers to adequate vitamin D synthesis.

Rural–urban comparison: why no significant difference?

Although we hypothesized a priori that rural patients might benefit from greater sun exposure, our results do not support a meaningful rural–urban gap in 25(OH)D. This null finding is itself informative. In the context of Assam, rural agricultural workers tend to cover most of their body with clothing as sun protection and practical field attire, limiting the exposed surface area available for cutaneous vitamin D synthesis. Urban patients, conversely, may have better access to fortified foods, dairy products, and vitamin D-containing supplements, partially offsetting their reduced outdoor activity. Additionally, the sample size of 40 patients while adequate for detecting the strong Vitamin D–disease activity correlation may have been underpowered to detect a modest rural–urban difference in 25(OH)D. A larger multicentre study would be needed to conclusively resolve this question.

Vitamin D and disease activity: a robust inverse relationship

Notwithstanding the null rural–urban finding, our data reveal a striking and consistent inverse association between 25(OH)D and all disease activity measures. The correlation with BASDAI ($r_s=-0.814$) and CRP ($r_s=-0.872$) are among the strongest reported in AS literature. This is biologically coherent: 1,25(OH)₂D, the

active form of vitamin D, suppresses Th17 differentiation and reduces IL-17A and IL-23 production the very cytokines that drive enthesal and axial inflammation in AS.^{5,13} Vitamin D also promotes VDR-mediated inhibition of NF- κ B signalling, decreasing TNF- α output. In our data, patients with vitamin D deficiency had a mean BASDAI of 6.3 compared to 3.7 in the normal group, and all 18 vitamin-D-deficient patients had active disease (BASDAI ≥ 4).

These findings align with a seminal Turkish study by Mermerci Başkan et al., who reported significantly higher BASDAI and BASFI scores in vitamin D-deficient AS patients and with a 2020 meta-analysis by Bai et al. confirming that lower 25(OH)D independently predicted higher BASDAI in pooled analysis ($\beta=-0.18$, 95% CI: −0.31 to −0.05).^{14,15} Our correlation coefficients exceed those reported in most individual studies, possibly because our cohort has a higher proportion of deficient patients and a wider range of disease activity, amplifying the observable gradient.

Clinical implications

The high prevalence of vitamin D deficiency across both rural and urban patients, combined with its strong association with disease activity, argues strongly for routine 25(OH)D monitoring in all AS patients attending AMCH and comparable centres in Northeast India. This is especially relevant given that vitamin D supplementation is safe, inexpensive, and widely available. Whether correction of deficiency directly reduces disease activity in AS remains a subject of ongoing randomized trials; however, the observational signal from our study and existing literature is compelling enough to support prophylactic supplementation as an adjunct to standard DMARDs and biologics.¹⁶

Limitations

Several limitations should be acknowledged. First, the cross-sectional design precludes causal inference lower vitamin D could be a consequence rather than a cause of higher inflammatory burden (through increased vitamin D catabolism by activated immune cells). Second, our sample of 40 patients from a single centre limits statistical power for subgroup analyses and generalizability. Third, seasonal variation in 25(OH)D was not controlled for, though data collection was conducted across multiple seasons. Fourth, HLA-B27 status a major genetic modifier of disease activity was not uniformly available for all patients and was therefore not included as a covariate. Fifth, dietary vitamin D intake, physical activity, and outdoor work hours were not quantitatively captured, limiting our ability to explain the lack of a rural–urban difference.

CONCLUSION

In this cohort of 40 AS patients from a tertiary centre in Assam, rural and urban residence did not significantly

predict differences in serum vitamin D status or disease activity. Both groups bore a substantial burden of vitamin D deficiency (collectively $\geq 77.5\%$ deficient or insufficient) and high disease activity (77.5% BASDAI-active). Crucially, serum 25(OH)D was strongly and inversely correlated with BASDAI, ASDAS-ESR, ASDAS-CRP, CRP, and ESR across the full cohort, indicating that vitamin D deficiency regardless of residential background tracks closely with disease severity.

These findings have practical implications: vitamin D screening should be incorporated into the routine workup of all AS patients in Northeast India, and deficiency should be corrected proactively. Larger multicentre longitudinal studies are needed to confirm or refute the rural–urban hypothesis with adequate power, determine whether vitamin D supplementation modifies disease trajectory in AS, and characterize the contribution of seasonal and behavioural determinants to the unexpectedly uniform vitamin D burden across residential categories in this region.

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