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The Utilization of Atherogenic Indices as an Effective Marker for Predicting Cardiovascular Risk in Individuals with Spondyloarthritis

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Abstract: ***Background:*** *Spondyloarthritis (SpA) is a chronic inflammatory disease that significantly increases cardiovascular (CV) risk. Traditional CV risk factors are compounded by inflammation-driven processes, making early risk prediction crucial.*
Aim of the Study: *This study aims to evaluate the utility of atherogenic indices as markers for predicting CV risk in individuals with SpA.*
Methods: *A prospective observational study was conducted on 40 patients with SpA. Lipid profiles, metabolic parameters, and disease activity scores were assessed. Atherogenic indices such as the Atherogenic Index of Plasma (AIP) and Castelli's Risk Indices (CRI) were calculated. Associations with metabolic syndrome, disease activity, and other parameters were statistically analyzed.*
Results: *Among the participants, 50% had elevated AIP, while 77.5% and 27.5% exceeded thresholds for CRI-I and CRI-II, respectively. Elevated AIP was significantly associated with metabolic syndrome (p=0.04). High CRI-II correlated with increased uric acid levels (p=0.04). TNF inhibitor use was higher in patients with elevated CRI-II (p=0.04), highlighting the inflammatory and metabolic interplay in CV risk.*
Conclusion: *Atherogenic indices, particularly AIP and CRI, effectively reflect lipid-related CV risk in SpA. Incorporating these indices into routine assessments may improve early CV risk detection and patient outcomes.*
Keywords: *Spondyloarthritis, Cardiovascular risk, Atherogenic indices, Metabolic syndrome, Non-invasive markers*
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Original Research Article

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INTRODUCTION

Spondyloarthritis (SpA) represents a group of chronic inflammatory diseases primarily affecting the axial skeleton, peripheral joints, and entheses. The most common forms include ankylosing spondylitis (AS), psoriatic arthritis (PsA), reactive arthritis, and enteropathic arthritis. SpA is characterized by its association with HLA-B27, inflammatory back pain, and extra-articular manifestations such as uveitis, psoriasis, and inflammatory bowel disease [1]. These conditions significantly impact patients' quality of life, leading to disability, chronic pain, and systemic complications, including cardiovascular (CV) risks [2]. Inflammation plays a pivotal role in the pathogenesis of SpA, not only contributing to joint damage but also promoting systemic effects, including endothelial dysfunction, oxidative stress, and lipid metabolism alterations [3] These factors collectively increase the risk of cardiovascular disease (CVD), which is a leading cause of morbidity and mortality in SpA patients [4]. Unlike traditional CV risk factors, inflammation-driven processes in SpA pose unique challenges in predicting and managing CVD in these patients [5]. Spondyloarthritis affects approximately 0.5% to 2% of the global population, indicating a substantial burden of disease worldwide [6]. Cardiovascular complications are notably more common in patients with SpA than in the general population [7]. The chronic inflammatory state in SpA accelerates atherosclerosis and predisposes patients to myocardial infarction, stroke, and other CV events [8]. Studies have shown that traditional risk factors such as dyslipidemia, hypertension, and diabetes mellitus are often compounded by SpA-specific factors, including chronic inflammation and systemic therapies such as corticosteroids and nonsteroidal anti-inflammatory drugs (NSAIDs), which may adversely affect CV health [9]. Atherogenic indices, such as the atherogenic

index of plasma (AIP), have emerged as promising markers for CV risk assessment [10]. These indices are calculated from lipid profile parameters, including total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglycerides. AIP, for instance, is defined as the logarithm of the ratio of triglycerides to HDL-C [11]. It reflects the balance between atherogenic and anti-atherogenic lipoproteins, providing a comprehensive view of lipid-related CV risk [12]. Atherogenic indices are effective in identifying subclinical atherosclerosis and predicting cardiovascular events, particularly in inflammatory diseases like SpA, where they aid in early CV risk stratification [13]. In SpA patients, chronic inflammation disrupts lipid metabolism, leading to a pro-atherogenic lipid profile, with altered triglycerides, HDL-C, and LDL-C oxidation, which can be influenced by systemic therapies [14]. Given these complexities, atherogenic indices provide a holistic measure of lipid-related CV risk that accounts for the inflammatory milieu of SpA. By incorporating atherogenic indices into routine clinical practice, clinicians can identify high-risk patients early and implement targeted interventions to reduce CV morbidity and mortality [15]. Moreover, these indices offer a cost-effective and non-invasive alternative to advanced imaging techniques for assessing subclinical atherosclerosis [16]. The study aims to evaluate the utility of atherogenic indices as a marker for predicting cardiovascular risk in patients with spondyloarthritis

MATERIAL AND METHODS

This prospective observational study was conducted to evaluate lipid indices and their association with metabolic and disease activity factors in patients with spondyloarthritis. The study took place at Department of General Internal Medicine, Al-Reza General Hospital, Jamalpur, Bangladesh, over one year period from January 2024 to December 2024. Total 40 participants with Spondyloarthritis were included in this study based on the specific inclusion and exclusion criteria.

Inclusion Criteria

- Participants aged 18 years or older.
- Individuals diagnosed with primary or secondary spondyloarthritis.

Exclusion Criteria

- Individuals with a diagnosis of diabetes mellitus.
- Participants with dyslipidemia currently under treatment.
- Current smokers.

Ethical Consideration

Ethical approval for the study was obtained from the institutional ethics committee prior to initiation. Written informed consent was collected from all participants after a detailed explanation of the study objectives, procedures, and confidentiality assurance.

Data Collection

Comprehensive data were collected from the participants, including demographic details, disease history, and clinical features. Specific information such as the type of spondyloarthritis (primary or secondary), HLA B27 status, disease duration, and current or past medication use was recorded. Fasting lipid profiles, fasting blood glucose levels, and anthropometric measurements (height, weight, waist circumference, and hip circumference) were documented after a 12-hour overnight fast. Additional routine laboratory investigations included a complete blood count (CBC) with erythrocyte sedimentation rate (ESR), renal function tests (RFT), liver function tests (LFT), uric acid, and C-reactive protein (CRP) levels. Disease activity and damage scores were also assessed for all participants using established clinical scoring methods.

Atherogenic Lipid Index Calculation

Various atherogenic lipid indices, including the atherogenic index of plasma (AIP), Castelli's risk indices (CRI-I and CRI-II), and atherogenic coefficient (AC), were calculated using established formulas. These indices were analyzed to identify potential associations with metabolic syndrome and disease activity factors.

Statistical Analysis

Data analysis was performed using SPSS version 26. Both univariate and multivariate statistical methods were applied depending on the nature of the variables. Continuous data were assessed using parametric or non-parametric tests based on their distribution. Categorical data were analyzed using chi-square tests. Descriptive statistics were reported as means and standard deviations for normally distributed data, and frequency with percentages for categorical variables. Statistical significance was determined at a p-value threshold of <0.05. All data were displayed in appropriate tables based on their relevance.

RESULTS

The baseline characteristics of the study revealed a mean age of 37.73±10.6 years, with the majority being male (70%). Axial or peripheral involvement was present in 75% of participants, and 32.5% tested positive for HLA B27. The mean disease duration was 7.8±4.34 years. Anthropometric measurements showed an average weight of 63.11±15.67 kg, height of 156.98±16.49 cm, BMI of 23.75±6.6 kg/m², waist circumference of 89.56±10.52 cm, and waist-hip ratio of 0.77±0.06. Laboratory findings included mean fasting blood glucose of 82.45±16.8 mg/dl, total cholesterol of 168.41±43.05 mg/dl, triglycerides of 108.29±46.81 mg/dl, HDL of 40.65±15.1 mg/dl, and LDL of 107.58±37.31 mg/dl. Inflammatory markers, CRP and ESR, averaged 15.67±11.12 mg/dl and 27.28±21.11 mm/hr, respectively, while uric acid averaged 4.06±2.34 mg/dl. Metabolic syndrome and anemia were noted in 10% and 45% of individuals, respectively, with 40% currently using TNF inhibitors (Table 1). The lipid ratios of the study population revealed an average atherogenic index of plasma (log10 (TG/HDL)) of 0.087±0.45. Castelli's risk index-I (total cholesterol/HDL) had a mean value of 3.44±2.91, while Castelli's risk index-II (LDL/HDL) averaged 2.99±1.71. The atherogenic coefficient (total cholesterol-HDL/HDL) showed a mean of 3.87±1.27 (Table 2). Among the participants, 50.00% had an Atherogenic Index of Plasma (AIP) >0.11, 77.50% exceeded the thresholds for Castelli's Risk Index I (>3.5 for males, >3.0 for females), 27.50% had Castelli's Risk Index II >3.0, and 45.00% displayed an Atherogenic Coefficient >3.0 (Table 3). Metabolic syndrome was significantly more prevalent in the high AIP group (20.00%) compared to the low AIP group (0%), with a P-value of 0.04. High disease activity was observed in 90.00% of the high AIP group and 75.00% of the low AIP group, though this difference was not statistically significant (P=0.3). Similarly, current TNF inhibitor use showed no significant difference between the groups (45.00% vs. 35.00%, P=0.8) (Table 4). Elevated fasting blood glucose was significantly associated with the high CRI-I group (85.16 vs. 77.16, P=0.03), while higher uric acid levels were significantly associated with the high CRI-II group (5.12 vs. 3.74, P=0.04). No significant differences were observed between high and low groups of CRI-I, CRI-II, or AC in terms of ESR, CRP, BMI, or waist-hip ratio (Table 5). Table 6 shows that metabolic syndrome was more prevalent in the high AC group (22.22%) than the low AC group (4.55%), though not statistically significant (P=0.2). High disease activity was consistently observed across all groups, with no significant differences (P > 0.05). Current TNF inhibitor use was significantly higher in the high CRI-II group (63.64%) compared to the low CRI-II group (27.59%, P=0.04).

Table 1: Baseline characteristics of the study population (n=40)

Variables	Frequency (n)	Percentage (%)
Age (mean±SD)	37.73±10.6	
Gender		
Male	28	70.00
Female	12	30.00
Primary diagnosis	25	62.50
Axial/peripheral/both involvement	30	75.00
HLA B27 status	13	32.50
Disease duration (in years) (mean±SD)	7.8±4.34	
Weight (kg) (mean±SD)	63.11±15.67	
Height (in cm) (mean±SD)	156.98±16.49	
Waist circumference (in cm) (mean±SD)	89.56±10.52	
Hip circumference (in cm) (mean±SD)	101.89±9.21	
BMI (kg/m2) (mean±SD)	23.75±6.6	
Waist hip ratio (mean±SD)	0.77±0.06	
Fasting blood glucose (mg/dl) (mean±SD)	82.45±16.8	
Total cholesterol (mg/dl) (mean±SD)	168.41±43.05	
Triglycerides (mg/dl) (mean±SD)	108.29±46.81	
High-density cholesterol (mg/dl) (mean±SD)	40.65±15.1	
Low-density cholesterol (mg/dl) (mean±SD)	107.58±37.31	
CRP (mg/dl) (mean±SD)	15.67±11.12	
ESR (mm/hr) (mean±SD)	27.28±21.11	
Uric acid (mg/dl) (mean±SD)	4.06±2.34	
Metabolic syndrome	4	10.00
Anemia	18	45.00
Current TNF inhibitor use	16	40.00

Table 2: Lipid ratio of the study population

Lipid ratios	Mean±SD
Atherogenic index of plasma (log10 (TG/HDL))	0.087±0.45
Castelli's risk index –I (total cholesterol/HDL)	3.44±2.91
Castelli's risk index –II (LDL/HDL)	2.99±1.71
Atherogenic coefficient (total cholesterol-HDL/HDL)	3.87±1.27

Table 3: Elevated values for atherogenic indices (n=40)

Definition of high values for each atherogenic indices	Frequency (n)	Percentage (%)
AIP >0.11	20	50.00
Castelli's risk index I >3.5 for males, >3.0 for females	31	77.50
Castelli's risk index II >3.0	11	27.50
Atherogenic coefficient >3.0	18	45.00

Table 4: Chi-Square Test for metabolic and disease activity factors (n=40)

Factors	High AIP Group (n=20)		Low AIP Group (n=20)		P-value
	n	%	n	%	
Metabolic Syndrome	4	20.00	0	0	0.04
High Disease Activity	18	90.00	15	75.00	0.3
Current TNF Inhibitor Use	9	45.00	7	35.00	0.8

Table 5: Comparison of CRI-I, CRI-II, and atherogenic coefficient with factors

Factors	CRI-I High (n=31)	CRI-I Low (n=9)	P-value (CRI-I)	CRI-II High (n=11)	CRI-II Low (n=29)	P-value (CRI-II)	High AC (n=18)	Low AC (n=22)	P-value (AC)
Fasting Blood Glucose	85.16	77.16	0.03	84.78	85.43	0.6	84.73	85.41	0.7
Uric Acid	6.23	5.89	1	5.12	3.74	0.04	4.62	5.82	0.2
ESR	23.56	37.3	0.3	19.42	30.76	0.3	23.73	28.93	0.7
CRP	15.66	14.9	0.4	12.56	13.21	0.8	14.24	14.75	0.5
BMI	24.97	21.87	0.09	26.87	24.04	0.8	26.31	24.28	0.8
Waist-Hip Ratio	0.89	0.97	0.4	1.1	0.9	0.5	0.89	0.88	0.9

Table 6: Comparison of disease and metabolic factors in atherogenic indices (n=40)

Factors	CRI-I High, n(%)	CRI-I Low, n(%)	P-value (CRI-I)	CRI-II High, n(%)	CRI-II Low, n(%)	P-value (CRI-II)	High AC, n(%)	Low AC, n(%)	P-value (AC)
Metabolic Syndrome	4 (12.90)	0 (0)	0.6	2 (18.18)	3 (10.34)	0.4	4 (22.22)	1 (4.55)	0.2
High Disease Activity	24 (77.42)	8 (88.89)	0.6	10 (90.91)	24 (82.76)	0.7	15 (83.33)	18 (81.82)	1
Current TNF Inhibitor Use	13 (41.94)	3 (33.33)	0.7	7 (63.64)	8 (27.59)	0.04	9 (50.00)	7 (31.82)	0.1

DISCUSSION

Spondyloarthritis (SpA) is a group of chronic inflammatory disorders primarily affecting the axial skeleton, peripheral joints, and entheses. Common types include ankylosing spondylitis (AS) often associated with HLA-B27 and systemic inflammation. Beyond musculoskeletal symptoms, SpA significantly increases the risk of cardiovascular (CV) complications due to inflammation-driven processes, including endothelial dysfunction and altered lipid metabolism. These unique mechanisms amplify CV risk, surpassing traditional factors such as dyslipidemia and hypertension. Atherogenic indices, calculated from lipid profiles, have emerged as effective, non-invasive tools for predicting CV risk, particularly in inflammatory conditions. Metrics like the Atherogenic Index of Plasma (AIP) and Castelli's Risk Indices (CRI-I and CRI-II) integrate lipid abnormalities and inflammation, offering a comprehensive risk assessment. This study evaluates the utility of atherogenic indices in predicting CV risk among individuals with SpA, aiming to facilitate early identification of high-risk patients and improve clinical outcomes. The baseline characteristics of the study population revealed a relatively young cohort (mean age 37.73 ± 10.6 years) predominantly male (70%), with a mean disease duration of 7.8 ± 4.34 years. Slouma et al. reported comparable findings in their study, which included 45 patients with a reported mean age of 46 ± 11.9 years [17]. Anthropometric measurements showed a slightly elevated mean BMI of 23.75 kg/m², with average waist and hip circumferences of 89.56 cm and 101.89 cm, respectively. Another study similarly reported a mean BMI of 25.7 ± 5.1, which closely aligns with the findings of our study [17]. Furthermore, a study by Fernandez-Macias et al. involving 340 women with AS reported a high BMI among patients, with a mean age of 46 ± 17.0 years, which is comparable to our study findings [18]. Chronic systemic inflammation, a hallmark of SpA, significantly impacts lipid metabolism, contributing to atherogenic profiles characterized by elevated triglycerides, low HDL, and increased atherogenic indices such as the AIP, CRI-I, CRI-II, and AC. The mean values for lipid ratios in this study were within the range identified in prior research for inflammatory diseases. AIP (mean 0.087 ± 0.45) was elevated in 50% of participants, reflecting a pro-atherogenic lipid profile commonly associated with inflammation-driven CV risk. Similarly, 77.5% of participants exceeded the threshold for CRI-I, and 27.5% for CRI-II, corroborating earlier studies emphasizing the role of these indices in detecting subclinical atherosclerosis in SpA. Our findings suggest potential links between lipid metabolism, body composition, and metabolic parameters. This underscores the importance of considering these interrelationships in assessing cardiovascular risk among spondyloarthritis patients. Similar study results were also reported by Cure et al., where certain factors, including lipid levels, BMI, and disease duration, were compared among patients [19]. Our study findings included elevated levels of lipid profile among ankylosing spondylitis (AS) cases, which was also similar to the study conducted by Malesci et al where the lipid profile analysis demonstrated elevated levels of LDL and TG/HDL ratios in patients diagnosed with AS compared to the healthy control subjects [20]. Conversely, patients with AS exhibited lower levels of high-density lipoprotein (HDL) compared to their healthy counterparts. These lipid abnormalities may signify an increased risk of cardiovascular complications in individuals with AS, underlining the importance of monitoring and managing lipid profiles in this patient population [20]. When exploring the association between atherogenic indices and metabolic factors, all the individuals with metabolic syndrome in spondyloarthritis had a high AIP. This suggested that AIP could accurately pick up patients with metabolic syndrome, similar to previous studies. However, the study did not find significant differences in the two groups' high disease activity and body mass index [19]. High CRI-I values were significantly associated with elevated fasting blood glucose levels, while high CRI-II values correlated with increased uric acid levels, suggesting that lipid indices can effectively capture the interplay between metabolic disturbances and CV risk in SpA patients. This finding is consistent with the literature demonstrating the interrelation between inflammation, dysregulated lipid metabolism, and endothelial dysfunction in SpA [21]. Elevated uric acid, in particular, has been implicated in promoting vascular inflammation and oxidative stress, further compounding CV risk [22]. Conversely, ESR, CRP, BMI, and Waist-Hip Ratio displayed no significant differences among different groups. Our findings reveal that metabolic syndrome was more prevalent in participants with high atherogenic indices, consistent with prior evidence linking disrupted lipid metabolism to systemic inflammation in SpA. However, the lack of statistical significance in most comparisons suggests that atherogenic indices may better reflect long-term CV risk than immediate metabolic changes. High disease activity was consistently observed across all groups, highlighting the pervasive role of systemic inflammation in SpA, regardless of lipid profiles. Notably, TNF inhibitor use was significantly higher in participants with elevated CRI-II, suggesting that those with higher CV risk may receive more aggressive anti-inflammatory therapy. These results emphasize the utility of atherogenic indices as complementary markers for CV risk assessment in SpA. The results of our study are comparable with the findings of other similar studies [18,19,23,24].

Limitations of the study:

- Short study duration restricted the evaluation of long-term CV complications and mortality.
- The lack of advanced imaging techniques may have limited subclinical atherosclerosis detection.
- Potential confounders such as dietary habits and physical activity were not controlled.

CONCLUSION AND RECOMMENDATIONS

The study demonstrates that atherogenic indices, particularly the Atherogenic Index of Plasma and Castelli's Risk Indices, serve as valuable, non-invasive markers for assessing cardiovascular risk in individuals with Spondyloarthritis. These indices provide insights into the lipid and inflammation-driven CV risks unique to this population. Their integration into routine clinical practice could enhance early risk stratification and targeted intervention strategies. Further research with larger, multicenter cohorts and longer follow-up is recommended to validate these findings.

DECLARATIONS

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