Section: General Medicine

Original Research Article

COMPARATIVE EVALUATION OF CARDIOVASCULAR RISK PROFILE IN PATIENTS OF RHEUMATOID ARTHRITIS AND HEALTHY CONTROLS AT A TERTIARY CARE HOSPITAL

Pragya Mishra[1], Aditi Sen[2], Sukriti Azad[2]

[1]*Assistant Professor, Department of General Medicine, Hind Institute of Medical Sciences, Barabanki, Lucknow, Uttar Pradesh, India.*
[2]*Assistant Professor, Department of General Medicine, Rajshree Medical Research Institute and Hospital, Bareilly, Uttar Pradesh, India.*

Received *: 12/04/2026*
Received in revised form *: 05/05/2026*
Accepted *: 27/05/2026*

Corresponding Author:
Dr. Sukriti Azad,
Assistant Professor, Department of General Medicine, Rajshree Medical Research Institute and Hospital, Bareilly, Uttar Pradesh, India..
Email: sukritiazad@gmail.com

DOI: 10.70034/ijmedph.2026.2.596

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (2); 3615-3622

ABSTRACT

Background: Rheumatoid arthritis is a chronic systemic inflammatory disorder associated with an increased risk of cardiovascular morbidity and mortality. In addition to traditional cardiovascular risk factors, persistent inflammation, altered lipid metabolism, reduced physical activity, and disease-related vascular changes may contribute to accelerated atherosclerosis in these patients. The aim is to comparatively evaluate cardiovascular risk profile in patients of rheumatoid arthritis and healthy controls at a tertiary care hospital.

Materials and Methods: This hospital-based, observational, cross-sectional comparative study included 104 participants, comprising 52 patients with rheumatoid arthritis and 52 age- and sex-matched healthy controls. Demographic characteristics, anthropometric measurements, conventional cardiovascular risk factors, blood pressure, glycaemic indices, lipid profile, inflammatory markers, electrocardiographic and echocardiographic findings, carotid intima-media thickness, carotid plaques, and Framingham Risk Scores were assessed.

Results: The mean age was comparable between rheumatoid arthritis patients and controls (49.60 ± 10.20 versus 49.10 ± 9.80 years; p=0.799). Rheumatoid arthritis patients had significantly higher body mass index, waist circumference, and waist-to-hip ratio. Hypertension, physical inactivity, central obesity, dyslipidaemia, and metabolic syndrome were significantly more frequent in the rheumatoid arthritis group. Systolic and diastolic blood pressure, fasting plasma glucose, glycated haemoglobin, triglycerides, LDL cholesterol, non-HDL cholesterol, total cholesterol/HDL ratio, and atherogenic index of plasma were significantly higher, whereas HDL cholesterol was significantly lower among rheumatoid arthritis patients. Mean carotid intima-media thickness was significantly greater in rheumatoid arthritis patients than controls (0.74 ± 0.14 versus 0.63 ± 0.11 mm; p<0.001), and carotid plaques were more frequent (26.92% versus 9.62%; p=0.022). The mean Framingham Risk Score was also significantly higher in rheumatoid arthritis patients (12.80 ± 8.60% versus 8.10 ± 6.40%; p=0.002).

Conclusion: Patients with rheumatoid arthritis had a significantly greater burden of conventional cardiovascular risk factors, systemic inflammation, subclinical atherosclerosis, and predicted cardiovascular risk than matched healthy controls. Routine cardiovascular screening and early preventive intervention should be incorporated into comprehensive rheumatoid arthritis management.

Keywords: Rheumatoid Arthritis; Cardiovascular Risk; Dyslipidaemia; Carotid Intima-Media Thickness; Framingham Risk Score.

INTRODUCTION

Rheumatoid arthritis is a chronic, systemic autoimmune inflammatory disorder primarily characterised by persistent synovitis, progressive cartilage destruction, bone erosion and varying degrees of functional disability. Although articular manifestations dominate its clinical presentation, rheumatoid arthritis affects several extra-articular organs and is associated with important long-term comorbidities. Cardiovascular disease is among the most clinically significant of these complications because it contributes substantially to premature morbidity and mortality. The cardiovascular burden associated with rheumatoid arthritis includes coronary artery disease, myocardial infarction, heart failure, cerebrovascular disease, peripheral arterial disease and sudden cardiac death. Consequently, contemporary rheumatoid arthritis care requires attention not only to the control of joint inflammation but also to the early identification and management of cardiovascular risk.[1]

The excess cardiovascular risk associated with rheumatoid arthritis is multifactorial. Traditional risk factors such as hypertension, diabetes mellitus, smoking, obesity, physical inactivity and dyslipidaemia remain important; however, they do not completely explain the cardiovascular burden observed in this population. Rheumatoid arthritis-specific factors, including persistent systemic inflammation, longer disease duration, high disease activity, seropositivity, functional limitation and exposure to certain medications, may act independently or interact with conventional risk factors. This interaction creates a complex clinical profile in which apparently modest abnormalities in blood pressure, glucose metabolism or lipid levels may coexist with an enhanced inflammatory and proatherogenic state.[2] Chronic inflammation provides a major biological link between rheumatoid arthritis and accelerated atherosclerosis. Pro-inflammatory cytokines, altered immune-cell activity, oxidative stress and endothelial dysfunction can promote vascular injury, lipid modification, plaque formation and plaque instability. Inflammation may also influence insulin sensitivity, body composition, coagulation and vascular tone. These mechanisms support the view that rheumatoid arthritis and atherosclerotic cardiovascular disease share several immunological and inflammatory pathways. The cumulative inflammatory burden may therefore be relevant even before clinically apparent cardiovascular disease develops, making assessment of inflammatory activity an important component of cardiovascular evaluation in patients with rheumatoid arthritis.[3]

Lipid abnormalities in rheumatoid arthritis require careful interpretation because active inflammation may alter total cholesterol, low-density lipoprotein cholesterol and high-density lipoprotein cholesterol in ways that are not fully captured by conventional thresholds. Lipoprotein composition and function may become abnormal even when absolute lipid concentrations do not appear markedly elevated. Atherogenic ratios, non-high-density lipoprotein cholesterol and other composite lipid indices may therefore provide additional information. Similarly, anthropometric measures such as body mass index, waist circumference and waist-to-hip ratio may help identify general and central adiposity, both of which can contribute to metabolic syndrome and cardiovascular risk.[4]

The assessment of cardiovascular risk in rheumatoid arthritis remains challenging. Risk calculators developed for the general population may underestimate risk because most do not directly incorporate disease activity, systemic inflammation, autoantibody status, functional disability or cumulative treatment exposure. Differences among available risk-assessment tools can also lead to inconsistent classification. A comprehensive assessment should therefore combine calculated cardiovascular risk with clinical evaluation of conventional risk factors, rheumatoid arthritis characteristics and evidence of target-organ or vascular involvement. Regular screening is particularly important because cardiovascular risk factors may remain underdiagnosed or undertreated when clinical attention is focused mainly on musculoskeletal symptoms.[5]

Subclinical cardiovascular abnormalities may precede symptomatic disease. Resting electrocardiography and echocardiography can identify rhythm disturbances, ventricular hypertrophy, diastolic dysfunction and other structural or functional abnormalities. Carotid ultrasonography provides a non-invasive method for measuring carotid intima-media thickness and detecting atherosclerotic plaques, thereby offering direct information about vascular disease that may not be reflected adequately in conventional risk scores. Emerging biomarkers related to vascular inflammation, myocardial stress, endothelial dysfunction and immune activation are also being investigated as possible tools for refining cardiovascular risk stratification in rheumatoid arthritis. However, their routine clinical application requires further validation and standardisation.[6]

MATERIALS AND METHODS

This hospital-based, observational, cross-sectional study was designed to comparatively evaluate cardiovascular risk profile in patients of rheumatoid arthritis and age- and sex-matched apparently healthy controls at a tertiary care hospital. A total of 104 participants were included in the study. The study population was divided into two groups, with 52 patients diagnosed with rheumatoid arthritis constituting the study group and 52 age- and sex-matched apparently healthy individuals constituting the control group. Rheumatoid arthritis patients

attending the medicine and rheumatology outpatient or inpatient services were enrolled consecutively. Healthy controls were selected from hospital staff, attendants of patients, and individuals attending routine health check-up services who did not have rheumatoid arthritis or any other chronic inflammatory disease. The diagnosis of rheumatoid arthritis was established according to the 2010 American College of Rheumatology/European Alliance of Associations for Rheumatology classification criteria. These criteria included the number and site of involved joints, serological status for rheumatoid factor and anti-cyclic citrullinated peptide antibodies, acute-phase reactants, and duration of symptoms. Only patients with an established diagnosis of rheumatoid arthritis were included in the study group.

Inclusion Criteria

The study group included adult patients aged 18 years or above with a confirmed diagnosis of rheumatoid arthritis who provided written informed consent. Patients receiving disease-modifying antirheumatic drugs, corticosteroids, non-steroidal anti-inflammatory drugs, or biological therapy were also eligible for inclusion, and details of treatment were recorded. The control group included apparently healthy individuals aged 18 years or above who were matched with rheumatoid arthritis patients for age and sex and who provided written informed consent.

Exclusion Criteria

Participants with a history of acute infection, other autoimmune or chronic inflammatory diseases, malignancy, chronic liver disease, advanced chronic kidney disease, pregnancy, or lactation were excluded. Individuals receiving lipid-lowering therapy solely for a recently diagnosed lipid abnormality were also excluded when pretreatment lipid values were unavailable. Patients with acute cardiovascular events, including acute myocardial infarction, unstable angina, acute heart failure, or stroke at the time of recruitment, were excluded because these conditions could alter inflammatory markers and biochemical parameters. Controls with rheumatoid arthritis, connective tissue disease, or any clinically evident inflammatory disorder were not included.

Methodology

A predesigned and pretested data collection form was used to record demographic, clinical, laboratory, and treatment-related information. Demographic variables included age, sex, place of residence, occupation, and socioeconomic status. A detailed history was obtained regarding smoking, alcohol consumption, physical activity, dietary habits, hypertension, diabetes mellitus, dyslipidaemia, previous cardiovascular disease, cerebrovascular disease, peripheral arterial disease, and family history of premature cardiovascular disease. In female participants, menopausal status was also recorded.

Assessment of Rheumatoid Arthritis Characteristics

For rheumatoid arthritis patients, information regarding age at disease onset, disease duration, duration of morning stiffness, pattern of joint involvement, presence of deformities, extra-articular manifestations, and functional limitations was documented. Current and previous treatment with conventional disease-modifying antirheumatic drugs, biological agents, Janus kinase inhibitors, corticosteroids, non-steroidal anti-inflammatory drugs, and other relevant medications was recorded. The cumulative duration and current dose of corticosteroid therapy were noted wherever available.

Assessment of Disease Activity

Disease activity in rheumatoid arthritis patients was assessed using the Disease Activity Score in 28 joints based on erythrocyte sedimentation rate, designated as DAS28-ESR. Tender and swollen joint counts were assessed in 28 joints, and the patient's global health assessment was recorded using a visual analogue scale. Based on the DAS28-ESR value, disease activity was classified as remission, low, moderate, or high. Functional status was assessed using the Health Assessment Questionnaire–Disability Index wherever feasible.

Anthropometric Assessment

Body weight was measured to the nearest 0.1 kg using a calibrated digital weighing scale, with the participant wearing light clothing and no footwear. Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer. Body mass index was calculated as weight in kilograms divided by the square of height in metres and expressed as kg/m^2. Waist circumference was measured midway between the lower margin of the last palpable rib and the upper border of the iliac crest at the end of normal expiration. Hip circumference was measured at the widest portion of the buttocks, and the waist-to-hip ratio was calculated. Central obesity was assessed according to accepted cut-off values for the study population.

Blood Pressure Measurement

Blood pressure was measured using a validated sphygmomanometer after the participant had rested in a seated position for at least five minutes. An appropriately sized cuff was applied to the upper arm, and blood pressure was measured in both arms during the initial assessment. Two readings were obtained at an interval of at least five minutes, and the average value was used for analysis. Hypertension was defined based on a documented previous diagnosis, current use of antihypertensive medication, or persistently elevated blood pressure detected during the study assessment.

Laboratory Investigations

Venous blood samples were collected under aseptic precautions after an overnight fast. The investigations included complete blood count, fasting plasma glucose, glycated haemoglobin, serum urea, serum creatinine, liver function tests, serum uric acid, and thyroid-stimulating hormone where clinically indicated. The fasting lipid profile included total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol. Very-low-density lipoprotein cholesterol and non-

high-density lipoprotein cholesterol were calculated using standard methods, wherever applicable.

Assessment of Inflammatory and Rheumatological Markers

Inflammatory activity was assessed using erythrocyte sedimentation rate and C-reactive protein. High-sensitivity C-reactive protein was measured where facilities were available to assess low-grade systemic inflammation related to cardiovascular risk. Rheumatoid factor and anti-cyclic citrullinated peptide antibodies were measured in rheumatoid arthritis patients. Seropositivity and antibody titres were recorded and analysed in relation to cardiovascular risk factors and disease activity.

Assessment of Lipid-Related Cardiovascular Risk

In addition to individual lipid parameters, lipid ratios associated with cardiovascular risk were calculated. These included the total cholesterol-to-high-density lipoprotein cholesterol ratio, low-density lipoprotein cholesterol-to-high-density lipoprotein cholesterol ratio, triglyceride-to-high-density lipoprotein cholesterol ratio, and atherogenic index of plasma. The atherogenic index of plasma was calculated as the logarithm of the ratio of triglycerides to high-density lipoprotein cholesterol, with both values expressed in molar concentrations. Non-high-density lipoprotein cholesterol was calculated by subtracting high-density lipoprotein cholesterol from total cholesterol.

Assessment of Metabolic Risk Factors

Diabetes mellitus was identified on the basis of a previous physician diagnosis, use of glucose-lowering medication, fasting plasma glucose, or glycated haemoglobin values meeting accepted diagnostic criteria. Metabolic syndrome was assessed using standard criteria based on waist circumference, blood pressure, fasting glucose, triglyceride level, and high-density lipoprotein cholesterol level. The presence and number of metabolic syndrome components were compared between the rheumatoid arthritis and control groups.

Cardiovascular Examination

A detailed cardiovascular examination was performed in all participants. Pulse rate, rhythm, peripheral pulses, jugular venous pressure, cardiac apex, heart sounds, murmurs, and signs of heart failure or peripheral vascular disease were assessed. Participants were examined for carotid bruit, peripheral oedema, and evidence of impaired peripheral circulation. A resting 12-lead electrocardiogram was obtained to identify rhythm disturbances, conduction abnormalities, previous silent myocardial infarction, left ventricular hypertrophy, and ST–T wave changes.

Echocardiographic Assessment

Two-dimensional transthoracic echocardiography with Doppler assessment was performed where available using a standard echocardiography machine. Parameters included left ventricular dimensions, left ventricular ejection fraction, regional wall motion abnormalities, left ventricular hypertrophy, diastolic function, valvular abnormalities, pulmonary artery pressure, and pericardial effusion. Echocardiographic findings were interpreted by an experienced physician or cardiologist who was unaware of the participant's cardiovascular risk categorisation whenever feasible.

Assessment of Subclinical Atherosclerosis

Carotid ultrasonography was performed where facilities were available to assess carotid intima-media thickness and the presence of atherosclerotic plaques. Measurements were obtained from the far wall of the common carotid arteries using a high-frequency linear transducer. The mean of measurements obtained from both sides was used for analysis. A focal increase in carotid wall thickness or a clearly defined protrusion into the arterial lumen was considered suggestive of carotid plaque according to standard ultrasonographic criteria.

Cardiovascular Risk Estimation

The 10-year risk of developing a cardiovascular event was estimated using a validated cardiovascular risk assessment tool, such as the Framingham Risk Score. Variables used for risk estimation included age, sex, total cholesterol, high-density lipoprotein cholesterol, systolic blood pressure, treatment for hypertension, smoking status, and diabetes mellitus. Participants were categorised into low-, intermediate-, and high-risk groups according to the calculated score. The risk score was calculated for both rheumatoid arthritis patients and controls to permit direct comparison.

Statistical Analysis

Data were entered into a spreadsheet, checked for completeness, and analysed using IBM Statistical Package for the Social Sciences, version 27.0. Continuous variables were expressed as mean with standard deviation for normally distributed data and as median with interquartile range for non-normally distributed data. Categorical variables were expressed as frequencies and percentages. The normality of continuous variables was assessed using the Shapiro–Wilk test and graphical methods.

RESULTS

A total of 104 participants were enrolled in the study, comprising 52 patients with rheumatoid arthritis and 52 age- and sex-matched healthy controls.

The mean age of participants in the rheumatoid arthritis group was 49.60 ± 10.20 years, while that of the healthy control group was 49.10 ± 9.80 years. The difference was not statistically significant (p=0.799), indicating successful age matching between the two groups.

Similarly, both groups had identical gender distributions, with females constituting 76.92% and males 23.08% of participants, demonstrating effective sex matching (p=1.000). However, significant differences were observed in anthropometric measurements. The mean body mass index was significantly higher among rheumatoid arthritis patients (25.80 ± 4.10 kg/m²) compared to healthy controls (24.20 ± 3.60 kg/m²) (p=0.037).

Similarly, waist circumference was significantly greater in the rheumatoid arthritis group (88.60 ± 11.70 cm) than in controls (83.40 ± 10.20 cm) (p=0.017). The waist-to-hip ratio was also significantly elevated among rheumatoid arthritis patients (0.89 ± 0.08 versus 0.85 ± 0.07; p=0.008). [Table 1]

The prevalence of conventional cardiovascular risk factors was generally higher among rheumatoid arthritis patients than healthy controls. Hypertension was observed in 34.62% of rheumatoid arthritis patients compared to 17.31% of controls, and this difference was statistically significant (p=0.044). Diabetes mellitus was present in 23.08% of rheumatoid arthritis patients and 9.62% of controls. Although the prevalence was more than twice as high in the rheumatoid arthritis group, the difference did not achieve statistical significance (p=0.063). Dyslipidaemia was identified in 57.69% of rheumatoid arthritis patients compared to 34.62% of healthy controls, representing a statistically significant difference (p=0.018). Likewise, metabolic syndrome was present in 46.15% of rheumatoid arthritis patients and 23.08% of controls, demonstrating a significantly higher burden in the rheumatoid arthritis group (p=0.013). [Table 2]

The rheumatoid arthritis group demonstrated significantly higher mean systolic blood pressure (132.60 ± 16.80 mmHg) compared with controls (124.10 ± 13.70 mmHg) (p=0.006). Mean diastolic blood pressure was also significantly elevated among rheumatoid arthritis patients (82.80 ± 9.50 mmHg versus 78.60 ± 8.70 mmHg; p=0.021).

Markers of glucose metabolism were significantly worse in the rheumatoid arthritis group. Mean fasting plasma glucose was 104.80 ± 22.60 mg/dL among rheumatoid arthritis patients compared with 96.20 ± 14.80 mg/dL among controls (p=0.024). Similarly, glycated haemoglobin levels were significantly higher in rheumatoid arthritis patients (5.93 ± 0.86%) than controls (5.57 ± 0.54%) (p=0.012), indicating a less favourable glycaemic profile. [Table 3]

The mean disease duration among rheumatoid arthritis patients was 7.82 ± 5.14 years, indicating that most participants had established disease. The average duration of morning stiffness was 58.40 ± 26.30 minutes, reflecting active inflammatory joint involvement. The mean DAS28-ESR score was 4.63 ± 1.21, indicating moderate overall disease activity. Disease activity classification showed that only 7.69% of patients were in remission, while 13.46% had low disease activity. Nearly half of the patients (46.15%) had moderate disease activity, and 32.69% had high disease activity. Serological evaluation revealed rheumatoid factor positivity in 75.00% of patients and anti-cyclic citrullinated peptide antibody positivity in 78.85%, indicating a predominantly seropositive disease population. Joint deformities were observed in 30.77% of patients, suggesting chronic structural joint damage. Extra-articular manifestations were present in 23.08% of cases, demonstrating systemic disease involvement beyond the joints. With regard to treatment, 92.31% of patients were receiving conventional synthetic disease-modifying antirheumatic drugs, highlighting adherence to standard treatment protocols. Methotrexate was the most commonly used medication and was prescribed to 80.77% of patients. Corticosteroid therapy was being used by 57.69% of participants, whereas only 11.54% were receiving biological disease-modifying antirheumatic drugs or Janus kinase inhibitors. [Table 4]

Cardiovascular investigations demonstrated a significantly greater burden of cardiac abnormalities among rheumatoid arthritis patients. Electrocardiographic abnormalities were detected in 28.85% of rheumatoid arthritis patients compared with 11.54% of controls (p=0.028), indicating increased cardiac involvement.

Left ventricular hypertrophy was observed in 19.23% of rheumatoid arthritis patients and only 5.77% of controls, representing a statistically significant difference (p=0.038). Similarly, left ventricular diastolic dysfunction was significantly more common among rheumatoid arthritis patients (34.62%) than healthy controls (13.46%) (p=0.012). In contrast, left ventricular ejection fraction was comparable between groups (60.80 ± 5.60% versus 62.10 ± 4.80%; p=0.207), suggesting preserved systolic function in most participants.

Markers of subclinical atherosclerosis showed marked differences. The mean carotid intima-media thickness was significantly greater among rheumatoid arthritis patients (0.74 ± 0.14 mm) than controls (0.63 ± 0.11 mm) (p<0.001), indicating increased arterial wall thickening. Carotid atherosclerotic plaques were also significantly more prevalent in the rheumatoid arthritis group (26.92%) compared with controls (9.62%) (p=0.022), providing evidence of accelerated atherosclerotic changes.

The mean Framingham Risk Score was significantly higher among rheumatoid arthritis patients (12.80 ± 8.60%) than controls (8.10 ± 6.40%) (p=0.002), indicating an increased estimated 10-year cardiovascular risk.

Risk categorisation further demonstrated the adverse cardiovascular profile of rheumatoid arthritis patients. Low cardiovascular risk (<10%) was observed in only 53.85% of rheumatoid arthritis patients compared with 76.92% of controls. Intermediate cardiovascular risk (10–20%) was present in 28.85% of rheumatoid arthritis patients and 17.31% of controls, while high cardiovascular risk (>20%) was noted in 17.31% and 5.77%, respectively. The overall distribution of cardiovascular risk categories differed significantly between groups (p=0.037). Furthermore, when intermediate- and high-risk categories were combined, 46.15% of rheumatoid arthritis patients had an elevated cardiovascular risk compared with only 23.08% of healthy controls (p=0.013). [Table 5]

Table 1: Comparison of demographic and anthropometric characteristics between rheumatoid arthritis patients and healthy controls

Parameter	Rheumatoid arthritis group (n=52)	Healthy controls (n=52)	p-value
Age, years	49.60 ± 10.20	49.10 ± 9.80	0.799
Female sex	40 (76.92%)	40 (76.92%)	1.000
Male sex	12 (23.08%)	12 (23.08%)	1.000
Rural residence	30 (57.69%)	28 (53.85%)	0.693
Urban residence	22 (42.31%)	24 (46.15%)	0.693
Body mass index, kg/m²	25.80 ± 4.10	24.20 ± 3.60	0.037
Waist circumference, cm	88.60 ± 11.70	83.40 ± 10.20	0.017
Waist-to-hip ratio	0.89 ± 0.08	0.85 ± 0.07	0.008

Table 2: Comparison of conventional cardiovascular and metabolic risk factors

Cardiovascular risk factor	Rheumatoid arthritis group (n=52)	Healthy controls (n=52)	p-value
Hypertension	18 (34.62%)	9 (17.31%)	0.044
Diabetes mellitus	12 (23.08%)	5 (9.62%)	0.063
Current smoking	8 (15.38%)	6 (11.54%)	0.566
Family history of premature cardiovascular disease	10 (19.23%)	7 (13.46%)	0.426
Physical inactivity	31 (59.62%)	20 (38.46%)	0.031
Central obesity	28 (53.85%)	17 (32.69%)	0.029
Dyslipidaemia	30 (57.69%)	18 (34.62%)	0.018
Metabolic syndrome	24 (46.15%)	12 (23.08%)	0.013

Table 3: Comparison of blood pressure, glycaemic, lipid and inflammatory parameters

Parameter	Rheumatoid arthritis group (n=52)	Healthy controls (n=52)	p-value
Systolic blood pressure, mmHg	132.60 ± 16.80	124.10 ± 13.70	0.006
Diastolic blood pressure, mmHg	82.80 ± 9.50	78.60 ± 8.70	0.021
Fasting plasma glucose, mg/dL	104.80 ± 22.60	96.20 ± 14.80	0.024
Glycated haemoglobin, %	5.93 ± 0.86	5.57 ± 0.54	0.012
Total cholesterol, mg/dL	192.40 ± 39.60	178.60 ± 31.80	0.053
Triglycerides, mg/dL	162.70 ± 68.50	132.40 ± 49.70	0.011
HDL cholesterol, mg/dL	41.80 ± 9.60	47.20 ± 10.10	0.006
LDL cholesterol, mg/dL	118.90 ± 34.70	105.10 ± 29.50	0.031
Non-HDL cholesterol, mg/dL	150.60 ± 38.70	131.40 ± 32.50	0.007
Total cholesterol/HDL ratio	4.82 ± 1.31	3.92 ± 1.08	<0.001
Atherogenic index of plasma	0.22 ± 0.18	0.10 ± 0.16	<0.001
ESR, mm/hour	42.80 ± 21.60	16.30 ± 8.40	<0.001
C-reactive protein, mg/L	13.60 ± 10.90	3.80 ± 2.70	<0.001

Table 4: Clinical, serological and treatment profile of rheumatoid arthritis patients

Rheumatoid arthritis characteristic	Rheumatoid arthritis group (n=52)
Disease duration, years	7.82 ± 5.14
Morning stiffness duration, minutes	58.40 ± 26.30
DAS28-ESR score	4.63 ± 1.21
Disease remission	4 (7.69%)
Low disease activity	7 (13.46%)
Moderate disease activity	24 (46.15%)
High disease activity	17 (32.69%)
Rheumatoid factor positivity	39 (75.00%)
Anti-CCP antibody positivity	41 (78.85%)
Joint deformities	16 (30.77%)
Extra-articular manifestations	12 (23.08%)
Conventional synthetic DMARD use	48 (92.31%)
Methotrexate use	42 (80.77%)
Current corticosteroid use	30 (57.69%)
Biological DMARD or JAK inhibitor use	6 (11.54%)

Table 5: Comparison of cardiovascular investigations, subclinical atherosclerosis and cardiovascular risk categories

Cardiovascular parameter	Rheumatoid arthritis group (n=52)	Healthy controls (n=52)	p-value
Any electrocardiographic abnormality	15 (28.85%)	6 (11.54%)	0.028
Left ventricular hypertrophy	10 (19.23%)	3 (5.77%)	0.038
Left ventricular diastolic dysfunction	18 (34.62%)	7 (13.46%)	0.012
Left ventricular ejection fraction, %	60.80 ± 5.60	62.10 ± 4.80	0.207
Mean carotid intima-media thickness, mm	0.74 ± 0.14	0.63 ± 0.11	<0.001
Carotid atherosclerotic plaque	14 (26.92%)	5 (9.62%)	0.022
Framingham Risk Score, %	12.80 ± 8.60	8.10 ± 6.40	0.002
Low cardiovascular risk, <10%	28 (53.85%)	40 (76.92%)	0.037†
Intermediate cardiovascular risk, 10–20%	15 (28.85%)	9 (17.31%)	—
High cardiovascular risk, >20%	9 (17.31%)	3 (5.77%)	—
Combined intermediate or high risk, ≥10%	24 (46.15%)	12 (23.08%)	0.013

DISCUSSION

In the present study, the rheumatoid arthritis and control groups were well matched for age and sex, with mean ages of 49.60 ± 10.20 years and 49.10 ± 9.80 years, respectively, and identical female predominance of 76.92% in both groups. This comparability strengthens the interpretation that differences in cardiovascular risk were less likely to be due to age or sex imbalance. However, rheumatoid arthritis patients had significantly higher body mass index, waist circumference and waist-to-hip ratio than controls. The mean BMI was 25.80 ± 4.10 kg/m² in rheumatoid arthritis patients compared with 24.20 ± 3.60 kg/m² in controls, while waist circumference was 88.60 ± 11.70 cm versus 83.40 ± 10.20 cm and waist-to-hip ratio was 0.89 ± 0.08 versus 0.85 ± 0.07. These findings are comparable to the observations of Giles et al. (2010), who reported that abdominal fat distribution differed significantly in rheumatoid arthritis; although BMI and waist circumference were similar between rheumatoid arthritis patients and controls, visceral fat area was 45 cm² higher in men with rheumatoid arthritis and subcutaneous fat area was 119 cm² higher in women with rheumatoid arthritis. Thus, both the present study and Giles et al. support the concept that altered adiposity and central fat accumulation contribute to cardiometabolic risk in rheumatoid arthritis.[7]

The present study showed a higher burden of conventional cardiovascular risk factors in rheumatoid arthritis patients. Hypertension was significantly more common in rheumatoid arthritis patients than controls, 34.62% versus 17.31%, while diabetes mellitus was also more frequent, 23.08% versus 9.62%, although not statistically significant. Physical inactivity was significantly higher in rheumatoid arthritis patients, 59.62% versus 38.46%, and central obesity was also more frequent, 53.85% versus 32.69%. These findings are consistent with Chung et al. (2012), who compared 197 rheumatoid arthritis patients with 274 matched controls from the Multi-Ethnic Study of Atherosclerosis and reported that 80.00% of rheumatoid arthritis patients had at least one modifiable cardiovascular risk factor. In their study, hypertension was more common in rheumatoid arthritis patients than controls, 57.00% versus 42.00%, with p=0.001.[8]

Metabolic abnormalities were also more frequent in the rheumatoid arthritis group in the present study. Dyslipidaemia was present in 57.69% of rheumatoid arthritis patients compared with 34.62% of controls, and metabolic syndrome was present in 46.15% versus 23.08%, respectively. These results are in agreement with Rostom et al. (2013), who studied 120 rheumatoid arthritis patients and 100 age- and sex-matched controls and found that the frequency of metabolic syndrome in rheumatoid arthritis varied from 18.00% to 48.60% depending on the diagnostic criteria used, and was significantly higher than in controls for all definitions. The prevalence of metabolic syndrome in the present study, 46.15%, lies within the upper range reported by Rostom et al., suggesting that rheumatoid arthritis patients in the present hospital-based population had a marked metabolic risk burden. Rostom et al. also found that higher ESR and glucocorticoid use were independently associated with metabolic syndrome, which is relevant to the present study because ESR was markedly elevated and 57.69% of rheumatoid arthritis patients were receiving corticosteroids.[9]

The lipid and inflammatory profile in the present study further supports an increased atherogenic state among rheumatoid arthritis patients. Triglycerides were significantly higher in rheumatoid arthritis patients than controls, 162.70 ± 68.50 mg/dL versus 132.40 ± 49.70 mg/dL, while HDL cholesterol was significantly lower, 41.80 ± 9.60 mg/dL versus 47.20 ± 10.10 mg/dL. LDL cholesterol and non-HDL cholesterol were also significantly higher among rheumatoid arthritis patients. The total cholesterol/HDL ratio was 4.82 ± 1.31 in rheumatoid arthritis patients compared with 3.92 ± 1.08 in controls, and the atherogenic index of plasma was 0.22 ± 0.18 versus 0.10 ± 0.16. ESR and C-reactive protein were also significantly higher in rheumatoid arthritis patients, 42.80 ± 21.60 mm/hour and 13.60 ± 10.90 mg/L, respectively. Similar findings were reported by Dessie et al. (2020), who observed significantly higher total cholesterol, total cholesterol/HDL ratio, LDL/HDL ratio and high-sensitivity C-reactive protein in rheumatoid arthritis patients compared with controls, along with significantly lower HDL cholesterol. Therefore, the present findings support the view that dyslipidaemia in rheumatoid arthritis is closely linked with systemic inflammation and may contribute to accelerated atherosclerosis.[10] The clinical profile of rheumatoid arthritis patients in the present study showed established and active disease. Mean disease duration was 7.82 ± 5.14 years, mean morning stiffness duration was 58.40 ± 26.30 minutes and mean DAS28-ESR was 4.63 ± 1.21. Only 7.69% of patients were in remission, while 46.15% had moderate disease activity and 32.69% had high disease activity. Rheumatoid factor positivity was present in 75.00% and anti-CCP positivity in 78.85%, indicating a predominantly seropositive cohort. Yu et al. (2018), in the Chinese Registry of Rheumatoid Arthritis, reported 8071 registered rheumatoid arthritis patients with a female-to-male ratio of 4.03:1, remission by DAS28-CRP in only 14.88%, and among treatment-naïve patients, 38.84% had moderate and 38.11% had high disease activity.[11]

Cardiac evaluation in the present study demonstrated a higher frequency of abnormalities among rheumatoid arthritis patients. Any electrocardiographic abnormality was detected in 28.85% of rheumatoid arthritis patients compared with 11.54% of controls. Left ventricular hypertrophy was present in 19.23% versus 5.77%, and left ventricular diastolic dysfunction was present in 34.62% versus 13.46%, respectively. However, left ventricular ejection fraction was comparable

between groups, 60.80 ± 5.60% versus 62.10 ± 4.80%, suggesting preserved systolic function in most participants. These findings are comparable with Ghaleb et al. (2019), who studied 75 rheumatoid arthritis patients and 38 healthy controls and found left ventricular diastolic dysfunction in 37.30% of rheumatoid arthritis patients compared with 10.50% of controls. The close similarity between the present study's diastolic dysfunction rate of 34.62% and Ghaleb et al.'s 37.30% supports the importance of echocardiographic screening for early cardiac involvement in rheumatoid arthritis, even when systolic function is preserved.[12]

Subclinical atherosclerosis was significantly greater in rheumatoid arthritis patients in the present study. Mean carotid intima-media thickness was 0.74 ± 0.14 mm in rheumatoid arthritis patients compared with 0.63 ± 0.11 mm in controls, and carotid atherosclerotic plaques were present in 26.92% versus 9.62%, respectively. These findings are consistent with Mohan et al. (2014), who reported significantly higher mean carotid intima-media thickness in rheumatoid arthritis patients than controls, 0.598 ± 0.131 mm versus 0.501 ± 0.081 mm, with p=0.001, and carotid plaques in 15.60% of rheumatoid arthritis patients compared with 0.00% of controls. The present study showed higher absolute carotid intima-media thickness and plaque prevalence than Mohan et al., which may be explained by higher metabolic syndrome prevalence, greater dyslipidaemia, higher inflammatory markers and longer established disease in the present cohort. Both studies, however, strongly support the presence of accelerated subclinical atherosclerosis in rheumatoid arthritis.[13] The overall estimated cardiovascular risk was significantly higher in rheumatoid arthritis patients in the present study. The mean Framingham Risk Score was 12.80 ± 8.60% among rheumatoid arthritis patients compared with 8.10 ± 6.40% among controls. Low cardiovascular risk was observed in 53.85% of rheumatoid arthritis patients and 76.92% of controls, while intermediate or high cardiovascular risk was present in 46.15% of rheumatoid arthritis patients compared with 23.08% of controls. These results are highly comparable with Wagan et al. (2016), who studied 106 rheumatoid arthritis patients and 106 age- and sex-matched non-rheumatoid arthritis participants and reported a mean Framingham Risk Score of 12.90 ± 10.40% in rheumatoid arthritis patients compared with 8.90 ± 8.70% in non-rheumatoid arthritis controls, with p=0.001.[14]

CONCLUSION

The study demonstrated that patients with rheumatoid arthritis had a significantly greater burden of cardiovascular risk factors than age- and sex-matched healthy controls. They showed higher rates of hypertension, central obesity, dyslipidaemia, metabolic syndrome, adverse lipid indices, and systemic inflammation. Rheumatoid arthritis patients also had more frequent cardiac abnormalities, increased carotid intima-media thickness, carotid plaques, and higher Framingham Risk Scores. These findings support routine cardiovascular risk assessment and early preventive management as an essential component of rheumatoid arthritis care.

REFERENCES

1. DeMizio DJ, Geraldino-Pardilla LB. Autoimmunity and inflammation link to cardiovascular disease risk in rheumatoid arthritis. Rheumatol Ther. 2020;7(1):19–33. doi: 10.1007/s40744-019-00189-0.
2. Dijkshoorn B, Raadsen R, Nurmohamed MT. Cardiovascular disease risk in rheumatoid arthritis anno 2022. J Clin Med. 2022;11(10):2704. doi: 10.3390/jcm11102704.
3. Weber BN, Giles JT, Liao KP. Shared inflammatory pathways of rheumatoid arthritis and atherosclerotic cardiovascular disease. Nat Rev Rheumatol. 2023;19(7):417–428. doi: 10.1038/s41584-023-00969-7.
4. Popescu D, Rezus E, Badescu MC, Dima N, Seritean Isac PN, Dragoi IT, et al. Cardiovascular risk assessment in rheumatoid arthritis: accelerated atherosclerosis, new biomarkers, and the effects of biological therapy. Life (Basel). 2023;13(2):319. doi: 10.3390/life13020319.
5. Murphy L, Saab MM, Cornally N, McHugh S, Cotter P. Cardiovascular disease risk assessment in patients with rheumatoid arthritis: a scoping review. Clin Rheumatol. 2024;43(7):2187–2202. doi: 10.1007/s10067-024-06996-3.
6. Pamies A, Vallvé JC, Paredes S. New cardiovascular risk biomarkers in rheumatoid arthritis: implications and clinical utility—a narrative review. Biomedicines. 2025;13(4):870. doi: 10.3390/biomedicines13040870.
7. Giles JT, Allison M, Blumenthal RS, Post W, Gelber AC, Petri M, et al. Abdominal adiposity in rheumatoid arthritis: association with cardiometabolic risk factors and disease characteristics. Arthritis Rheum. 2010;62(11):3173–3182. doi: 10.1002/art.27629.
8. Chung CP, Giles JT, Petri M, Szklo M, Post W, Blumenthal RS, et al. Prevalence of traditional modifiable cardiovascular risk factors in patients with rheumatoid arthritis: comparison with control subjects from the Multi-Ethnic Study of Atherosclerosis. Semin Arthritis Rheum. 2012;41(4):535–544. doi: 10.1016/j.semarthrit.2011.07.004.
9. Rostom S, Mengat M, Lahlou R, Hari A, Bahiri R, Hajjaj-Hassouni N. Metabolic syndrome in rheumatoid arthritis: case control study. BMC Musculoskelet Disord. 2013;14:147. doi: 10.1186/1471-2474-14-147.
10. Dessie G, Tadesse Y, Demelash B, Genet S. Assessment of serum lipid profiles and high-sensitivity C-reactive protein among patients suffering from rheumatoid arthritis at Tikur Anbessa Specialized Hospital, Addis Ababa, Ethiopia: a cross-sectional study. Open Access Rheumatol. 2020;12:223–232. doi: 10.2147/OARRR.S264466.
11. Yu C, Li M, Duan X, Fang Y, Li Q, Wu R, et al. Chinese registry of rheumatoid arthritis (CREDIT): I. Introduction and prevalence of remission in Chinese patients with rheumatoid arthritis. Clin Exp Rheumatol. 2018;36(5):836–840.
12. Ghaleb RM, Abd Elazeem MI, Amin OA. Diastolic dysfunction in patients with rheumatoid arthritis. Egypt Rheumatol Rehabil. 2019;46:148–153. doi: 10.4103/err.err_6_19.
13. Mohan A, Sada S, Kumar BS, Sarma KVS, Devi BV, Rao PVLNS, et al. Subclinical atherosclerosis in patients with rheumatoid arthritis by utilizing carotid intima-media thickness as a surrogate marker. Indian J Med Res. 2014;140(3):379–386.
14. Wagan AA, Mahmud TEH, Rasheed A, Zafar ZA, Rehman AU, Ali A. Cardiovascular risk score in rheumatoid arthritis. Pak J Med Sci. 2016;32(3):534–538. doi: 10.12669/pjms.323.9779.