Wasit Journal for Pure Science

Journal Homepage: https://wjps.uowasit.edu.iq/index.php/wjps/index
e-ISSN: 2790-5241 p-ISSN: 2790-5233

The Impact of Rheumatoid Arthritis on Cardiovascular Health

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DOI: https://doi.org/10.31185/wjps.815
Received 10 April 2025; Accepted 09 June 2025; Available online 30 December 2025

ABSTRACT: Rheumatoid arthritis (RA) is a chronic systemic autoimmune disease characterized by persistent synovial inflammation that primarily targets the joints and can involve extra-articular organs, including the cardiovascular system. Although its precise etiology remains unclear, RA is believed to result from a complex interplay of genetic susceptibility (e.g., HLA-DR alleles), environmental factors, and microbial triggers. RA affects all age groups and both sexes, but it is approximately three times more prevalent in women than in men. Initial symptoms typically present as symmetric polyarthritis, affecting the small joints of the hands and feet, with progression to larger joints such as the knees and elbows. This study aimed to evaluate cardiovascular risk markers among RA patients in Wasit Governorate, Iraq. A total of 90 individuals aged 30–60 years were enrolled, comprising 60 RA patients (diagnosed based on clinical and laboratory criteria) and 30 age- and sex-matched healthy controls. Among RA patients, 73.3% were female, and the majority were either obese (70.0%) or overweight (21.7%). RA patients exhibited significantly elevated ESR values, along with variations in hematological indices such as reduced RBC count and hemoglobin levels. Moreover, serum lipid analysis revealed significantly elevated levels of TC, TG, LDL, and VLDL, accompanied by decreased HDL levels.

Keywords: Rheumatoid Arthritis, CBC, Cardiovascular Health, Lipid Profile,

1. INTRODUCTION

Rheumatoid arthritis (RA) is a chronic systemic autoimmune disease characterized by persistent synovial inflammation and potential multi-organ involvement. Individual differences in clinical presentation and disease severity can result in gradual joint degradation and extra-articular symptoms, which may include pulmonary involvement, subcutaneous nodules, and ocular inflammation, if left untreated, can cause lifelong disability or even death. Prevalence rates for RA have been shown to range from 0.24% to 2% worldwide [1]. It is still unclear what causes this illness. RA significantly impairs quality of life, as measured by tools like the Disease Activity Score-28 (DAS28) and the Health Assessment Questionnaire Disability Index (HAQ-DI) [2].

Patients with RA have significant disability, early unemployment, decreased life expectancy, and gradual joint degeneration, all of which have a detrimental effect on their health [3].

A chronic systemic inflammatory disease that mostly affects the joints but can spread to other body parts is RA. In addition to possible impacts on the pulmonary and circulatory systems, associated extra-articular symptoms include a markedly elevated risk of cardiovascular disease [4].

A chronic, systemic inflammatory illness, RA gradually impairs joint function and results in irreparable, lifelong impairment. Evidence indicates that RA patients have a much higher risk of cardiovascular illness, including a higher incidence of myocardial infarction and stroke, in addition to joint deterioration [5].

Chronic inflammation in RA contributes to endothelial dysfunction and a pro-atherogenic environment, mediated by cytokines such as IL-6 and TNF-α, thereby increasing the risk of myocardial infarction and stroke. Through their ability to reduce inflammation or their potential to raise heart risk, medications used to treat RA can either directly or indirectly affect heart health. The most commonly utilized drugs are glucocorticoids, JAK inhibitors, methylchloroquine, methotrexate, TNF-α inhibitors, and IL-6 antagonists (like tocilizumab) [6].

Patients with RA frequently get a complete blood count (CBC) to track changes linked to the disease's inflammatory activity and to assess medication side effects. Hematological abnormalities such as normocytic anemia, elevated platelet counts, reduced neutrophils and lymphocytes, and elevated monocytes are signs of systemic inflammation linked to RA [7].

A lipid profile is a pattern of fats in the blood. A lipid profile typically includes levels of total cholesterol (TC), triglycerides (TGs), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein cholesterol (VLDL-C)[8].

This study aimed to evaluate hematological and lipid profile alterations associated with increased cardiovascular risk in RA patients compared to healthy controls.

2. LITERATURE REVIEW

Many studies have examined the effects of RA on cardiovascular health. To ascertain the prevalence of dyslipidemia in RA patients, Erum et al. conducted an investigation. The "Rheumatology Clinic" at Jinnah Postgraduate Medical Center (JPMC), Karachi, was the site of this prospective, cross-sectional, observational study, which ran from November 2013 to May 2014. Two hundred participants with RA who were diagnosed using the 2010 ACR/EULAR criteria were included in the research. All patients had laboratory testing for creatinine, ALT, CBC, TSH, and fasting lipid profiles (LDL, HDL, and total cholesterol). There were 177 (88.5%) female patients and 23 (11.5%) male patients out of 200. The average length of the illness was 3.82±3.03 years, and the average age was 36.31±10.46 years. Of the 107 patients (53.5%) who had dyslipidemia, 83 (41.5%) had low HDL, the most frequent abnormality [9].

A Research by Kasem et al. examined alterations in lipid profiles in RA patients and assessed the relationship between lipid profiles and the severity of the disease. 150 patients were classified as having RA based on the 2010 ACR/EULAR criteria. Evaluations were conducted on demographic information, medical and treatment history, and history of arthritis. DAS28 (ESR) was used to assess disease activity. Immunological tests included anti-CCP, CRP, ESR, and RF.Levels of the lipid profile were measured. Patients with elevated TG were 38 (25.3%), those with elevated TC were 69 (46%) and those with reduced HDL-C were 85 (56.7%) [10].

Al-Fatlawi et al. conducted an investigation to determine the correlation between rheumatic arthritis and alterations in lipid profiles and anemia in both male and female patients in the Holy City of Karbala. Twenty healthy volunteers were split into two groups (10 men and 10 females each), and 20 patients were split into two groups (10 males and 10 females each). Hematological parameters and lipid profiles were assessed. The results indicated a substantial drop in HDL ($P\leq 0.04$) and a significant rise in TC ($P\leq 0.05$), LDL ($P\leq 0.02$), VLDL ($P\leq 0.02$), and TG ($P\leq 0.01$) in male RA patients. In female RA patients, there was a highly significant drop in HDL ($P\leq 0.01$) and a considerable rise in TC ($P\leq 0.05$) and LDL ($P\leq 0.01$). Data revealed a very significant rise ($P\leq 0.01$) in ESR for both male and female RA and a substantial drop ($P\leq 0.05$) in serum ferritin for male RA. The results showed that both male and female RA patients had significantly lower hemoglobin levels than the control group. The RBC count for female RA patients decreased significantly ($P\leq 0.01$), according to the results. Both male and female RA patients had significantly lower platelets and MCV, according to the study [11].

In order to evaluate the lipid profile, neutrophil-lymphocyte ratio (NLR), and platelet-lymphocyte ratio (PLR) in various rheumatic illnesses and investigate their relationship to disease activity and/or severity, Passant et al. conducted an investigation. 70 controls and 257 patients were enlisted, including 47 with RA, 100 with systemic lupus erythematosus (SLE), 49 with systemic sclerosis (SSc), 33 with axial spondyloarthritis (axSpA), and 28 with vasculitis (21 with primary vasculitis and 7 with Behçet's disease, or "BD"). Each disease's activity and/or severity were evaluated. TC, HDL, TG, LDL, VLDL, and the LDL:HDL ratio was all assessed as part of the lipid profile. Significantly increased NLR, PLR, and HDL were seen in RA ($p<0.0001$, $p=0.001$, $p=0.01$). The disease activity score (DAS28) had a negative correlation with NLR ($r=-0.3$, $p=0.02$) and was strongly linked to dyslipidemia ($p=0.02$). There was a strong correlation between TG and NLR and PLR ($p=0.02$, $p=0.03$). Significant increases in NLR, PLR, and TG were seen in SLE ($p<0.0001$, $p<0.0001$, $p<0.001$). NLR ($p=0.005$) and dyslipidemia ($p=0.01$) were substantially correlated with the SLE disease activity index (SLEDAI). The damage index and PLR had a negative relationship ($r=-0.2$, $p=0.01$). Significant correlations were seen between SLEDAI and TG ($r=0.4$, $p<0.0001$), LDL: HDL ($r=0.4$, $p<0.0001$), and HDL ($r=-0.4$, $p<0.0001$). NLR and PLR were much greater in SSc ($p<0.0001$, $p=0.03$). The modified Rodnan skin score (mRss) and HDL had a negative relationship ($r=-0.3$, $p=0.04$) [12].

3. STUDY FEATURES

3.1. Levels of some blood parameters in patients with RA

Patients with RA frequently get a complete blood count (CBC) to track changes linked to the disease's inflammatory activity and to assess medication side effects. Hematological abnormalities such as normocytic anemia, elevated platelet counts, reduced neutrophils and lymphocytes, and elevated monocytes are signs of systemic inflammation linked to RA [7].

A. White Blood Cells (WBC)

White blood cell (WBC) counts are crucial clinical tests for identifying and diagnosing diseases like viral or bacterial infections and autoimmune disorders like RA. WBCs migrate into the synovial membrane, causing inflammation that leads to joint pain, swelling, and progressive bone and cartilage erosion. This chronic inflammation thickens the normal synovial membrane, contributing to joint damage over time. Self-reactive B cells are a key factor in the disease's pathogenesis, producing autoantibodies that form immune complexes that accumulate in the joint and stimulate the infiltration of additional immune cells into the synovial tissue [13].

Leukocytes in RA impact not only the joints but also the cardiovascular system. Systemic inflammation from leukocyte activation promotes the development of atherosclerosis and is associated with an increased risk of coronary heart disease and heart failure. Neutrophil macrophages play a significant role in the formation of unstable plaques in the arteries, increasing the likelihood of heart attacks. Elevated levels of inflammatory mediators, such as IL-6 and TNF-α, associated with leukocyte activation contribute to endothelial dysfunction and increased cardiac fibrosis. Patients with RA also exhibit elevated levels of C-reactive protein (CRP), an inflammatory marker strongly associated with joint inflammation and cardiovascular events [14].

B. Red Blood Cells (RBC)

RA is a chronic inflammatory disease that affects joints and causes systemic disorders, including anemia as a common hematological complication. Anemia of chronic disease (ACD) is the most common among RA patients, attributed to persistent inflammation that increases the secretion of the hormone hepcidin, which reduces iron absorption and limits its availability for red blood cell production. A clinical analysis of RA patients found 37.3% to have ACD, 11.8% to have iron deficiency anemia (IDA), and 17.6% to have both types. Hepcidin concentrations were significantly higher in ACD patients, reinforcing the hypothesis of hepcidin's pivotal role in iron regulation during inflammation. Anemia in RA affects the cardiovascular system, causing decreased oxygen delivery to tissues, cardiac strain, and an increased risk of heart disease. Anemia is an indirect indicator of inflammation severity and disease progression in RA, and is associated with decreased physical performance and quality of life. Therefore, assessing red blood cell and iron stores is crucial in RA treatment plans to reduce cardiac complications and improve patients' quality of life [15].

C. Hemoglobin

Anemia, a common complication of RA, is caused by chronic inflammation factors like iron imbalance, impaired red blood cell formation, and impaired response to erythropoietin. Hemoglobin is a negative indicator of the inflammatory response in the acute phase but not included in composite activity indices like the DAS. Low hemoglobin levels in RA patients are associated with increased physical disability and may reflect clinical or subclinical inflammatory activity. However, the complexity of anemia in RA prevents its inclusion as a direct therapeutic target or a fixed component of disease management guidelines. Low hemoglobin levels in RA are also associated with an increased risk of cardiovascular disease, as inflammatory anemia promotes atherosclerosis and increases cardiac burden, making hemoglobin an indirect but important indicator in assessing cardiac risk in RA patients [16].

D. Hematocrit (HCT)

Hematocrit (HCT) is a vital indicator of red blood cell levels in the blood, indicating the blood's ability to transport oxygen to vital body tissues. It is crucial for diagnosing and monitoring various clinical conditions, including anemia, dehydration, and chronic diseases. In RA, low hematocrit levels are often observed due to chronic inflammatory anemia, a common symptom attributed to inflammatory cytokines like IL-6. Low hematocrit is an early sign of disease activity and may be accompanied by low hemoglobin, which can impair physical performance and increase fatigue. Low hematocrit is also associated with an increased risk of cardiovascular disease, especially in RA patients with chronic inflammation. Studies have shown that anemia, reflected in low hematocrit, increases the cardiac workload, leading to conditions like heart failure and myocardial infarction. Low hematocrit is also associated with an increased risk of cardiac death, even in those without clinically significant anemia. Although not currently used as indicators of RA activity, their low levels should be considered in overall patient assessment [17].

E. Platelet

RA is linked to an increased platelet count due to an inflammatory response. Platelets, which release cytokines like IL-6, TNF-α, and IL-1β, stimulate the production of larger platelets by activating thrombopoietin production. Inflammation affects platelet volume (MPV) and function, with increased or decreased MPV depending on disease severity and co-morbidities. Activated platelets release microparticles that increase inflammation and clotting. Platelet parameters, particularly MPV and PDW, are associated with RA activity, but results are conflicting. Increased platelet volume may lead to complications like metabolic syndrome and stroke. Chronic inflammation and platelet alterations increase the risk of atherosclerosis and heart disease in RA patients [18].

3.2. Lipid Profile

A. Total Cholesterol (TC)

Cholesterol is a crucial biological molecule with 27 carbon atoms and a hydroxyl group. It plays a vital role in cell membrane construction, providing stability and fluidity for their functions. Cholesterol is insoluble in water, so it binds to lipoproteins to form complex molecules that are transported through the bloodstream. Cell membranes in mammals are primarily dependent on cholesterol for maintaining structure and function. Inflammatory factors in RA disrupt the functions of adipose tissue, skeletal muscle, and other organs, leading to abnormalities in lipid levels, particularly HDL and LDL cholesterol concentrations. Dyslipidemia, a major contributor to atherosclerosis, is a key component in enhancing cardiovascular risk in RA patients [19].

In 2017, the European League Against Rheumatism (EULAR) recommended cardiovascular risk assessment in RA patients at least once every five years. Dyslipidemia occurs when plasma lipid levels change, with increased plasma cholesterol and triglycerides leading to atherosclerosis. A comprehensive understanding of lipid metabolism is essential to explain the physiological processes associated with lipid transport and function. Cholesterol is found in all animal tissues and as a component of living membranes, steroid hormones, and bile acids. Cholesterol combines with certain proteins to form a complex called lipoprotein in the bloodstream. LDL-C transports cholesterol from its production site in the liver to various body tissues, while HDL-C transports excess cholesterol to the liver. Cholesterol is an essential fat for the human body, and if it exceeds its needs, it can cause atherosclerosis, hypertension, and coronary heart disease [20].

B. Triglyceride (TG)

Glycerol, a key fat storage form, is found in HDL-C and LDL-C and is a major component of VLDL and chylomicrons. High triglyceride levels are a risk factor for coronary heart disease due to their role in atherosclerosis. Elevated triglyceride levels are observed in patients with RA, which is associated with atherosclerosis progression and heart disease complications [21].

C. High Density Lipoprotein Cholesterol (HDL-C)

High-density lipoprotein (HDL) is a protein found on lipoproteins, with proteins accounting for 50%, cholesterol 20%, and phospholipids and triglycerides 30%. HDL transports cholesterol from peripheral tissues to the liver, providing protection against atherosclerosis and supporting endothelial cell function. Patients with active RA have lower levels of total cholesterol and HDL cholesterol. HDL is crucial in assessing the risk of cardiovascular disease (CVD), with classic epidemiological studies showing an inverse relationship between HDL levels and CVD risk. Higher plasma HDL levels are associated with a significantly lower incidence of CVD events [22].

D. Cholesterol Low- Density Lipoprotein (LDL-C)

Low-density lipoprotein (LDL), also known as "bad cholesterol," is a significant factor in atherosclerosis and inflammation. Its structure, metabolism, and function are crucial for cardiovascular disease research and treatment strategies. In RA, elevated LDL-C levels may be due to the enzyme PCSK9, which promotes the degradation of LDL receptors and reduces LDL-C clearance, leading to its accumulation in plasma. Reducing LDL-C cholesterol levels can reduce the risk of CVD and lower mortality rates by 40%. LDL-C, which constitutes 7% of total blood cholesterol, is one of the most important types of cholesterol in terms of its pathological impact. Lowering LDL-C has been shown to reduce the risk of angina by approximately one-third and plays a significant role in coronary atherosclerosis. Understanding the structure, metabolism, and function of LDL is essential for advancing cardiovascular disease research and treatment strategies [23].

E. Very Low-Density Lipoprotein Cholesterol (VLDL-C)

Lipoprotein, a type of lipoprotein produced by the liver, has a molecular diameter of 30-80 nanometers and a density greater than chylomicrons. Its primary function is to transport phospholipids, cholesterol, and triglycerides throughout the body. Recent research indicates a close relationship between elevated very-low-density lipoprotein cholesterol (VLDL-C) levels and an increased risk of cardiovascular disease in patients with RA. RA patients have characteristic lipid abnormalities, including low total cholesterol, LDL-C, and HDL-C, along with elevated triglyceride and VLDL-C

levels. Chronic inflammation, known as the "lipid paradox," plays a major role in increasing this risk. Recent studies have shown residual cholesterol, including VLDL-C, is a strong and independent predictor of CVD risk in RA patients [24].

4. MATERIAL AND METHODS

4.1. Study design

Blood samples were collected from 90 patients, 60 men and 30 women, with rheumatoid arthritis (HRP) during the specified period from January 10, 2024, to January 1, 2025. In terms of age groups, 14 (15.6%) were under 40 years of age, 25 (27.7%) were between 40 and 49 years of age, and 51 (56.7%) were over 50 years of age. The RA patients included 8 (13.3%) people under the age of 40, 16 (26.7%) people between 40 and 49 years, and 36 (60.0%) people over the age of 50, while the control group included 6 (20.0%) people under the age of 40, 9 (30.0%) people between 40 and 49 years, and 15 (50.0%) people over the age of 50. In terms of age groups, there were 14 (15.6%) people under the age of 40, 25 (27.7%) people between 40 and 49 years, and 51 (56.7%) people over the age of 50. The RA patients included 8 (13.3%) people under the age of 40, 16 (26.7%) people between the ages of 40 and 49, and 36 (60.0%) people over the age of 50, while the control group included 6 (20.0%) people under the age of 40, 9 (30.0%) people between the ages of 40 and 49, and 15 (50.0%) people over the age of 50. The study was approved by the hospital administration, and informed consent was obtained from all participants.

4.2. Blood sample

Samples were collected between November 2024 and January 2025 from the Wasit Prosthetics Center, as well as Al-Karma and Al-Zahraa Teaching Hospitals. To collect 7 ml of venous blood, the procedure required obtaining information from both patients and healthy subjects, completing a form for each subject, and disinfecting the blood collection site with cotton and 70% diluted alcohol. The following materials and methods were used:

- 2 ml of blood were placed in ESR tubes to measure the erythrocyte sedimentation rate.
- 2 ml were placed in sodium citrate tubes to generate plasma after centrifugation for 15 minutes at 2,000 rpm to measure fibrinogen.
- To separate the clotted portion of the plasma from the blood, we placed the remaining 3 ml of blood in gelatin-filled glass tubes. The tubes were then left at room temperature for one to two hours to allow the globules to complete and coagulate.
- The samples were then centrifuged at 3,000 rpm for ten minutes.
- The serum is then collected using a micropipette into Eppendorf test tubes and stored at -20°C until ELISA is used for biochemical testing.

4.3. Statistical Analysis

SPSS 20 has been used to organize and analyze statistics. Comparison statistics (independent t-test, Mann-Whitney test t, and Pearson/Spearman bivariate correlations) and explanatory statistical metrics (frequency distribution and percentages displayed in tables and figures, along with mean ± s standard deviation) were used. Statistical significance was defined as a P-value of 0.05.

5. RESULTS AND DISCUSSION

5.1. Levels of some blood parameters in RA

Some blood parameters were compared in RA patients and healthy controls, and the results are shown in Table 1. The mean white blood cell count was 7.55 ± 1.9 and 7.00 ± 1.01 in RA patients and healthy controls, respectively; the level was higher in the patient group than in the healthy control group, but the difference was not statistically significant ($P = 0.160$). The mean red blood cell count was 4.64 ± 0.53 and 4.96 ± 0.61 in RA patients and healthy controls, respectively; the level was lower in the patient group than in the healthy control group, but the difference was statistically significant ($P = 0.018$). These findings are indicative of chronic low-grade inflammation impacting erythropoiesis.

The mean hemoglobin (Hb) levels were 12.56 ± 1.80 and 13.25 ± 1.48 in RA patients and healthy controls, respectively; The level was lower in the patient group compared to the healthy control group, but the difference was not statistically significant ($P = 0.069$).

Regarding the mean platelet count levels, the current results show that the mean platelet levels in RA patients were not statistically significantly higher than the mean platelet levels in the healthy control group, reaching 279.95 ± 36.96

versus 257.80 ± 24.46, respectively (P = 0.137). The current results also show a non-statistically significant difference between RA patients and the healthy control group according to all other criteria (P < 0.05).

Table 1. - Average levels of some blood parameters in RA patients and healthy controls.

	Case-control comparison		P-Value
	Patients	**Healthy (healthy sample)**	
	***n* = 60**	***n* = 30**	
White Blood Cells count			
Mean ± Standard Deviation	**7.55 ± 1.9**	**7.00 ± 1.01**	**0.16** †
Range	2.80 – 12.80	4.70- 8.70	**NS**
Red Blood Cells count (million/ µl)			
Mean ± Standard Deviation	**4.64 ± 0.53**	**4.96 ± 0.61**	**0.018** †
Range	3.73 – 6.20	3.83- 6.20	**S**
Hemoglobin (Hb) (g/dl)			
Mean ± Standard Deviation	**12.56 ± 1.80**	**13.25 ± 1.48**	**0.069** †
Range	8.70 -18.30	11.20-16.40	**NS**
Hematocrit (HCT)			
Mean ± Standard Deviation	**39.58 ± 5.47**	**37.88 ± 5.1**	**0.159** †
Range	27.70 -56.70	28.44-49.10	**NS**
Lymphocytes count			
Mean ± Standard Deviation	**2.46 ± 0.51**	**2.54 ± 0.55**	**0.61** †
Range	1.10 -3.80	1.10-3.90	**NS**
Lymphocytes percentage (%)			
Mean ± Standard Deviation	**34.40 ± 7.84**	**32.71 ± 6.14**	**0.851** †
Range	15.70 -52.20	20.00-39.00	**NS**
MXD level			
Mean ± Standard Deviation	**0.60 ± 0.12**	**0.62 ± 0.12**	**0.693** †
Range	0.16 -1.10	0.50-0.90	**NS**

MXD percentage (%)			
Mean ± Standard Deviation	**8.17 ± 2.90**	**8.04 ± 2.26**	**0.833** †
Range	3.0 -15.80	5.90-13.00	**NS**
Neutrophil count			
Mean ± Standard Deviation	**4.42 ± 0.91**	**4.08 ± 0.84**	**0.315** †
Range	1.30 -9.50	2.40-7.20	**NS**
Neutrophil percentage (%)			
Mean ± Standard Deviation	**58.78 ± 7.58**	**56.48 ± 4.31**	**0.213** †
Range	32.00 -76.00	49.20-69.10	**NS**
Platelet Count (thousand/ µl)			
Mean ± Standard Deviation	**279.95 ± 36.96**	**257.80 ± 24.46**	**0.137** †
Range	109.00 -507.00	205.00-338.00	**NS**

n: number of cases; SE: standard error; †: independent samples t-test; HS: Highly significant at P ≤ 0.001. NS: not significant at P > 0.05

The results of the current study show some differences between RA patients and the control group regarding basic hematological parameters, including red blood cell count, white blood cell count, hemoglobin, and platelets. The mean white blood cell count was higher in RA patients compared to healthy controls, but the difference was not statistically significant (P = 0.160). This trend is consistent with the literature indicating that chronic inflammation in RA can lead to a slight increase in white blood cell count, especially during periods of disease activity, as a result of immune activation [25].

The results also showed a statistically significant decrease in red blood cell count (P = 0.018) and a non-significant decrease in hemoglobin level (P = 0.069) in RA patients compared to healthy controls. This is consistent with the phenomenon of "anemia associated with chronic inflammation," which is common in RA and results from a disruption in the bone marrow response to erythropoietin, as well as iron sequestration within immune cells, this is consistent with [26].

The mean platelet count was higher in RA patients (279.95) compared to the control group (257.80), but the difference did not reach statistical significance (P = 0.137). However, Targońska-Stępniak et al. demonstrated that thrombocytosis could be a marker of inflammatory activity in RA, as platelets are affected by the secretion of IL-6 and TNF-α [27].

5.2. Results of lipid profile analysis (TC, TG, VLDL, LDL and HDL)

A comparison of lipid levels (TC, TG, VLDL, LDL, and HDL) between patients and the control group was performed, and the results are shown in Table (3-7). The mean serum TC levels were 232.65 ± 25.2 mg/dL and 178.14 ± 7.57 mg/dL in RA patients and the healthy group, respectively. The mean levels were higher in RA patients than in the healthy group, and the difference was significant (P = 0.011). The difference was highly statistically significant (P < 0.001).

The mean serum TG levels were 194.5 ± 9.31 mg/dL and 167.5 ± 6.72 mg/dL in RA patients and the healthy group, respectively. The mean levels were higher in the patient group than in the healthy group, and the difference was significant

(P = 0.011). The elevated TG levels observed (mean = 160 mg/dL, P < 0.01) may reflect the metabolic effects of chronic inflammation and corticosteroid use.

Regarding the mean serum VLDL levels, they were 39.9 ± 1.86 mg/dL and 33.49 ± 1.34 mg/dL in RA patients and healthy controls, respectively. The mean levels were higher in RA patients than in the control group, and the difference was highly statistically significant (P = 0.034).

As for the mean serum LDL levels, they were 169.6 ± 17.02 mg/dL and 106.7 ± 9.08 mg/dL in RA patients and healthy controls, respectively. The mean levels were higher in RA patients than in the healthy control group, and the difference was highly statistically significant (P < 0.001).

However, there was a significant decrease in the protein level HDL cholesterol in RA patients compared to the healthy group (P > 0.05). The significant reduction in HDL among RA patients supports the hypothesis that RA contributes to a pro-atherogenic lipid profile.

Table 2. - Results of lipid profile analysis (TC, TG, VLDL, LDL and HDL) in RA patients and healthy controls.

Groups	Chole	TG	VLDL	LDL	HDL
	(mg/dl)	**(mg/dl)**	(mg/dl)	**(mg/dl)**	**(mg/dl)**
RA patients	**232.65 ± 25.2**	**194.5 ± 9.31**	**39.9±** 1.86	**169.6±** 17.02	**22.59 ± 3.78**
	183.2-300.0	180.64-238.86	36.13-47.77	121.6-240.45	14.68-33.0
Healthy people	**178.14 ±** 7.57	**167.5 ±** 6.72	**33.49 ±** 1.34	**106.7 ±** 9.08	**35.93 ±** 5.96
	164.6-199.74	168.87-194.56	33.77-38.91	79.35-127.48	27.94-50.60
p-value	**0.001****	**0.011****	**0.034****	**0.001****	**0.001****

SD: standard deviation; †: Independent T test **: significant at P > 0.05

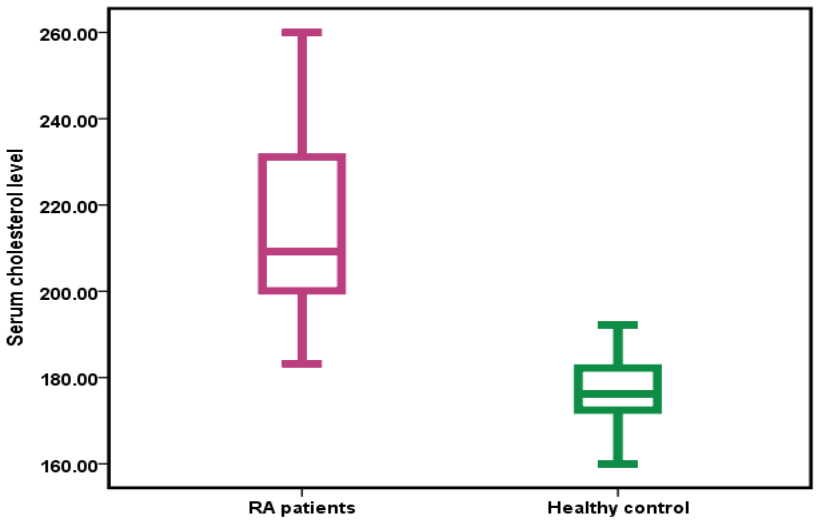

FIGURE 1. - Average serum cholesterol level in RA patients and healthy groups.

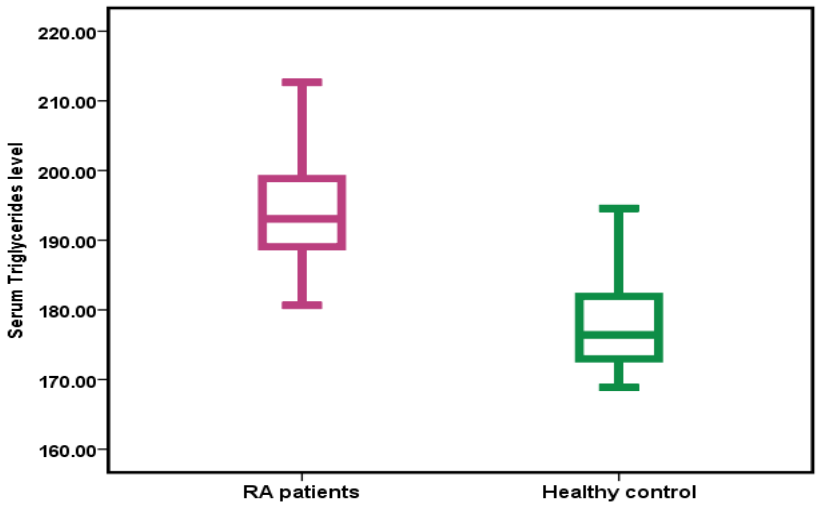

FIGURE 2. - Average serum triglyceride levels in patients and healthy controls

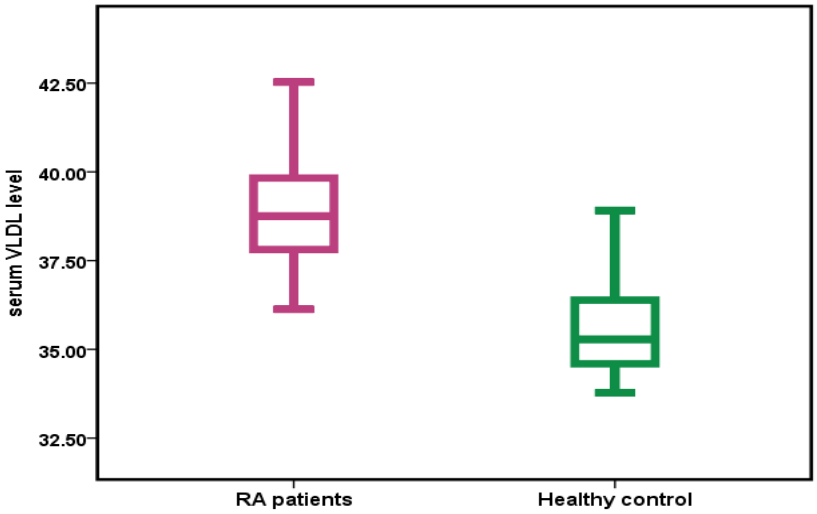

FIGURE 3. - Average serum VLDL levels in RA patients and healthy groups

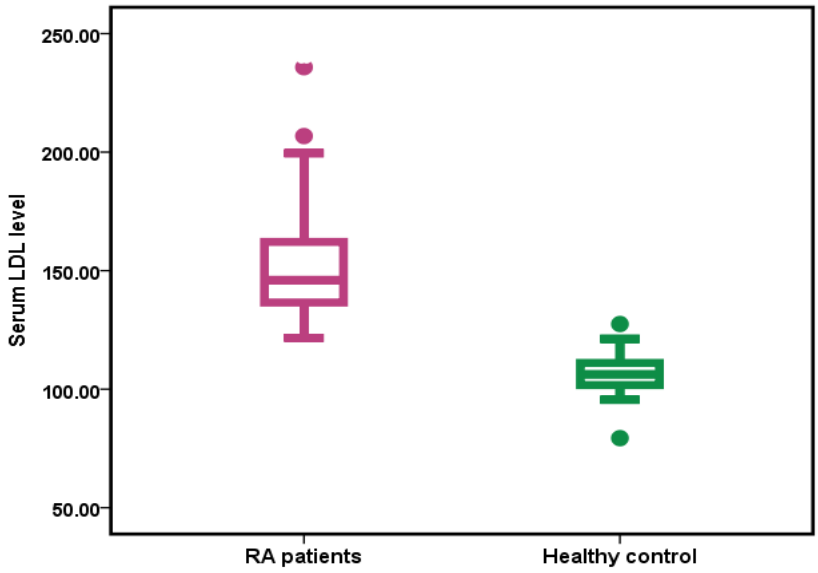

FIGURE 4. - Average serum LDL level in RA patients and healthy group.

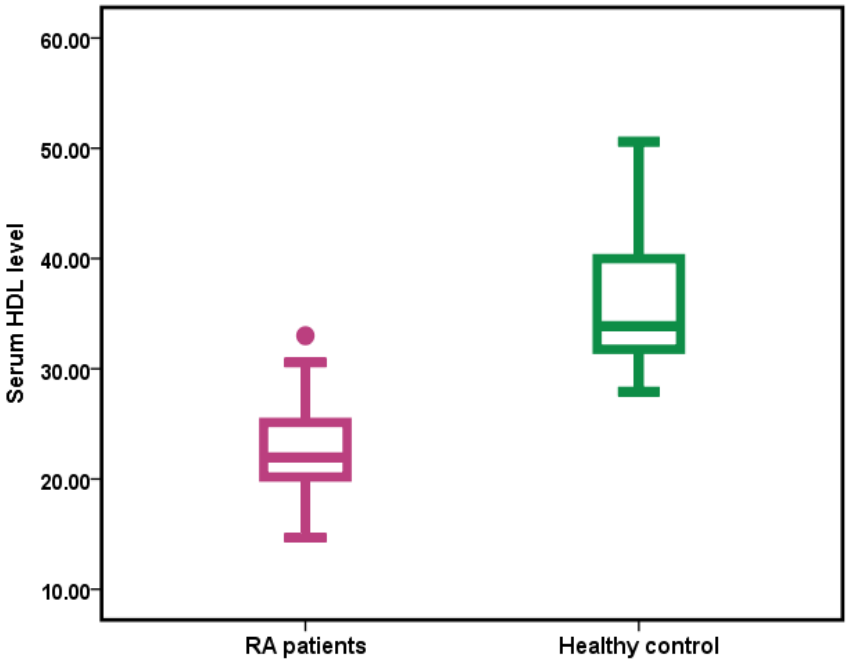

FIGURE 5. - Average serum HDL level in patients and healthy groups.

The study results showed that RA patients had higher levels of TG, TC, VLDL, and LDL, while significantly lower levels of high-density lipoprotein (HDL) were observed compared to the healthy control group. These differences were statistically significant for most indicators, reinforcing the notion of dyslipidemia associated with RA. These findings are consistent with several recent studies, including [28], which indicated that RA patients are prone to specific lipid abnormalities known as the "lipid paradox," whereby, despite elevated inflammatory markers, harmful lipids (LDL, VLDL, and TG) are elevated while HDL is decreased. A study by Gao et al. [29] demonstrated that chronic inflammation associated with RA increases triglyceride and LDL cholesterol levels via the influence of inflammatory cytokines on lipid metabolism, increasing the risk of cardiovascular disease.

The findings of decreased HDL in RA support the findings of Nurmohamed et al. [30], which demonstrated that persistent inflammation causes a decrease in functional HDL, leading to a loss of its vascular protective properties.

Thus, these findings reinforce the importance of routinely monitoring lipid profiles in RA patients, with a focus on reducing their cardiovascular risk by combining anti-inflammatory therapy with lipid management.

6. CONCLUSION

This study highlights the coexistence of anemia of chronic disease and atherogenic dyslipidemia in RA patients, which may elevate cardiovascular risk.

According to the study's findings, women were far more likely than males to have RA (73.3% versus 26.7%). Additionally, the data show that a minor fraction of RA patients in the research sample had normal weight (8.3%), with the majority being obese (70.0%) or overweight (21.7%).

Regarding hematological characteristics, the study's findings revealed that patients of both sexes had a significantly higher erythrocyte sedimentation rate (ESR) than healthy controls ($P = 0.001$). Basic hematological indicators, such as hemoglobin, platelets, white blood cell count, and red blood cell count, were also shown to differ between RA patients and the control group.

The study's findings also revealed that, in contrast to the healthy control group, RA patients had significantly lower levels of HDL and higher levels of TG, TC, VLDL, and LDL. For the majority of markers, these changes were statistically significant, confirming the idea that RA is linked to dyslipidemia.

According to the study's findings, individuals with RA have a number of risk factors, including obesity, dyslipidemia, high fibrinogen levels, chronic inflammation, and other biochemical and hematological indicators, that are both directly and indirectly related to heart disease. In order to lower the risk of future cardiac issues, routine cardiovascular risk assessment should be considered in RA management protocols, even in the absence of overt cardiac symptoms.

Further longitudinal studies incorporating inflammatory markers such as CRP, IL-6, and TNF-α are recommended to validate and expand upon these findings.

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