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Correlation Between Lipid Profile and Bacterial Infection in Iraqi Rheumatoid Arthritis Cases

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ABSTRACT:

Background: Rheumatoid arthritis (RA) is a chronic inflammatory disorder associated with dyslipidemia and increased cardiovascular risk. Bacterial infections may further exacerbate metabolic disturbances. This study evaluated correlated between lipid profile and bacterial infection in Iraqi R.A.

Methods: A case-control study was conducted with (No.=90) participants divided into three groups: Rheumatoid arthritis with bacterial infection (No.=30), R.A without bacterial infections (No.=30), and control (No.=30). Lipid profiles, inflammatory markers (ESR; Procalcitonin & CRP), and synovial fluid parameters were assessed. used culture and biochemical tests for identification bacteria.

Results: R.A patients demonstrated significant lipid alterations compared to control. The most changes occurred in patients with bacterial R.A, characterized by decreased total cholesterol (149.26 ± 23.52 mg/dL) and both (LDL, HDL) as (83.77 ± 16.95 mg/dL), (31.19 ± 6.72 mg/dL) respectively as well as elevated triglycerides (165.86 ± 33.04 mg/dL) ($p < 0.001$). negative correlated between lipid parameters and inflammatory markers ($p < 0.01$). so the predominant pathogens were both *Staphylococcus aureus* and *Escherichia coli* as (35.7 & 27.7) % respectively, Synovial fluid analysis showed significantly elevated WBC counts and LDH levels in infected joints ($p < 0.001$), whilst no significant differences in lipid profiles among different bacterial isolates.

Conclusions: Bacterial infection exacerbates lipid disturbances in R.A patients, likely driven by systemic inflammation. The inverse relationship between lipid levels and inflammatory markers supports the "lipid paradox" in RA. These findings highlight the importance of cardiovascular risk assessment in Rheumatoid arthritis patients with concurrent infections.

INTRODUCTION

Persistent synovial inflammation, increasing joint degeneration, and serious systemic consequences are the hallmarks of rheumatoid arthritis (RA), a chronic inflammatory illness (McInnes and Schett, 2011). Patients with RA often have dyslipidemia, with raised triglycerides and decreased HDL-C levels being the most common abnormality (Myasoedova *et al.*, 2011). The inflammatory-lipid interaction is seen in the inverse relationship between HDL-C and C-reactive protein (CRP) levels (Choy *et al.*, 2014). Triglycerides rise while total cholesterol, LDL-C, and HDL-C fall as a result of acute bacterial infections (Feingold and Grunfeld, 2000). similar to how dyslipidemia can be reversed when RA inflammation is managed (Toms *et al.*, 2010). Numerous arthritides are linked to bacterial infections; reactive Rheumatoid arthritis develops after gastrointestinal or genitourinary infections, whereas septic arthritis is caused by organisms such Staphylococcus aureus directly invading the joint (Mathews *et al.*, 2010). Additionally, by activating Toll-like receptors, bacterial lipopolysaccharide (LPS) may exacerbate chronic RA inflammation. (Abdollahi-Roodsaz *et al.*, 2008). Studies have looked at lipid profiles in inflammatory joint disorders in Iraq. Iraqi RA patients' lipid profiles and their relationship to inflammatory markers were assessed by Sulaiman *et al.*, 2023. In patients with active RA, Ali *et al.*, 2022 found significant decreases in total cholesterol, triglycerides, HDL and LDL, and which returned to normal following DMARD treatment. APOE genotypes and cardiovascular risk in Iraqi RA patients were examined by Khalil *et al.*, 2026. Galeefa *et al.*, 2015 identified *E. coli* as a trigger for reactive Rheumatoid arthritis, while Hussein *et al.*, 2023 showed that higher LPS levels correlated with disease activity in RA. Convergence of chronic Rheumatoid arthritis, bacterial infection, and altered lipid metabolism represents an important area of research. Understanding how bacterial infection affects lipid levels in Iraqi patients with RA can contribute to an understanding of disease mechanisms and the identification of cardiovascular risk factors (Zbala *et al.*, 2026). Current study aim to evaluate lipid levels and their relationship with bacterial infection in Iraqi patients with RA.

PATIENTS AND METHODS

Study Design: Case-control study conducted in Baghdad/Iraq.

Participants: 90 participants divided into: Group I: Rheumatoid arthritis with bacterial infection (n=30), Group II: Rheumatoid arthritis without bacterial infection (n=30) and Group III: controls (n=30)

Inclusion: Age 18-70 years, confirmed Rheumatoid arthritis diagnosis.

Exclusion: Chronic diseases(Pregnancy, antibiotic therapy within 48 hours, incomplete data, lipid-lowering therapy.

Sample Collection:

- Blood (5 mL): Serum for lipid profile and inflammatory markers, EDTA for ESR.
- Synovial fluid: For WBC count, biochemistry (glucose, LDH, lactate(Protein), Gram stain, and culture.

Laboratory Methods:

- Lipid profile (Total cholesterol;TG;LDL-Cand HDL-C) by enzymatic methods.
- ESR (Westergren), CRP (immunoturbidimetry)(Procalcitonin (immunochemiluminescence).
- Bacterial identification by culture and biochemical tests.

Clinical Assessment: DAS28 and CDAI scores for RA patients.

Statistical Analysis: SPSS v25. ANOVA, t-test, logistic regression ,Pearson correlation($P < 0.05$ significant).

Ethics: Institutional approval and written informed consent obtained.

RESULTS:

1. Clinical & Demographic Characteristics of Study groupPopulation

This study evaluated lipid profiles and bacterial infections status in 90 Iraqi participants, divided into three groups (No.= 30 for each group) : Rheumatoid arthritis with infections,(Group I),(Rheumatoid arthritis without bacterial infection), and control(Group III).

The clinical&demographic characteristics presented in Table 1 showed no statistically significant differences between the groups regarding sex ($p=0.860$);age ($p=0.723$);BMI ($p=0.417$) and disease duration ($p=0.362$). However, a statistically significant increase in inflammatory marker levels (ESR and CRP) was observed in both groups with Rheumatoid arthritis compared to the control group, with the elevated levels recorded in first group (ESR: 67.42 ± 21.19 mm/h; CRP: 52.63 ± 18.89 mg/L; $p<0.001$)

Table(1): Clinical &Demographic Characteristics of the Study Groups

Parameter (mean $\pm$ SD)	Group I	Group II	Group III	P-value
Age (years)	49.11 ± 10.99	50.27 ± 10.85	47.93 ± 12.16	0.723
Sex				0.860
Male	13 (46.0%)	11 (39.0%)	14 (45.3%)	
Female	17 (54.0%)	19 (61.0%)	16 (55.7%)	
Body Mass Index (BMI) (kg/m ²)	27.85 ± 3.50	27.10 ± 4.101	26.61 ± 3.85	0.417
Disease Duration (years)	3.85 ± 2.42	4.50 ± 3.12	-	0.362
ESR (mm/hr)	67.42 ± 21.19	53.80 ± 18.40	9.97 ± 3.49	<0.001*
CRP (mg/L)	52.63 ± 18.89	39.26 ± 16.71	3.45 ± 1.31	<0.001*
Procalcitonin (ng/mL)	3.89 ± 1.47	0.56 ± 0.39	0.09 ± 0.05	<0.001*

2. Lipid Profile Parameters Among the Study Groups

Results in table-2 showed significant differences ($p< 0.001$) for three groups regarding all lipid parameters, with the exception of the total cholesterol to TC/HDL ratio. Specifically, both total cholesterol and HDL, cholesterol exhibited a marked progressive increase from Group 1 to Group 3, whereas triglycerides and VLDL showed a marked progressive decrease across the groups. The highest levels of both LDL and non-HDL were recorded in Group-2 compared to the other two groups. However, CT/HDL ratio decreased gradually from Group 1 to Group 3, whilst this change wasn't statistically significant ($p = .168$).

Table(2): Lipid Profile Parameters for Study Groups

Parameter (mg/dL)	Group I	Group II	Group III	P-value
Total Cholesterol	149.26 ± 23.52	175.44 ± 27.72	197.64 ± 21.94	<0.001*
TG	165.86 ± 33.04	144.20 ± 27.52	123.72 ± 21.17	<0.001*
HDL	31.19 ± 6.72	37.62 ± 7.44	40.22 ± 8.32	<0.001*
Non-HDL	117.10 ± 22.48	138.69 ± 24.93	111.45 ± 24.71	<0.001*
LDL	83.77 ± 16.95	108.82 ± 21.35	98.49 ± 21.16	<0.001*
TC/HDL Ratio	4.91 ± 1.41	4.73 ± 1.20	4.26 ± 0.97	0.168
VLDL	33.96 ± 6.50	30.05 ± 5.72	25.91 ± 4.32	<0.001*

3. Synovial Fluid Biochemical Parameters

The results in Table 3 showed significant difference for parameters between first and second groups ($p < 0.001$). The first group showed a significant increase in white blood cell count ($67,419 \pm 23,160$ cells/mm³), lactate dehydrogenase (839.35 ± 216.58 units/L), and total protein (5.85 ± 1.27 g/dL), along with a significant decrease in glucose levels (32.45 ± 11.28 mg/dL).

Table(3): Synovial Fluid Biochemical Parameters for Study Groups

Parameter	Group I	Group II	P-value
WBC Count (cells/mm ³)	$67,419 \pm 23,160$	$13,390 \pm 5,72$	$<0.001^*$
Glucose (mg/dL)	32.45 ± 11.28	58.70 ± 15.11	$<0.001^*$
LDH (U/L)	839.35 ± 216.58	413.87 ± 129.65	$<0.001^*$
Total Protein (g/dL)	5.85 ± 1.27	4.31 ± 1.09	$<0.001^*$

Statistically significant ($P\text{-value} < 0.05$).

4. Bacterial Isolates

Current results showed in table-4 the most common bacteria was *Staphylococcus aureus* (33.34%), followed by *Escherichia coli* (30%), *Streptococcus spp.* (13.34%), *Klebsiella pneumoniae* (10.0%), and *Pseudomonas aeruginosa* (6.6%).

Table(4): Distribution of Bacterial Isolates in Patients with Septic Rheumatoid arthritis (n=30)

Bacterial Isolate	Number	Percentage (%)
<i>Staphylococcus aureus</i>	10	33.34
<i>Escherichia coli</i>	9	30.00
<i>Streptococcus spp.</i>	4	13.34
<i>Klebsiella pneumoniae</i>	3	10.00
<i>Pseudomonas aeruginosa</i>	2	6.66
<i>Enterococcus spp.</i>	1	3.33
<i>Proteus mirabilis</i>	1	3.33
Total	30	100%

5. Comparison of Lipid Profile According to Bacterial Isolate

The results in Table-5 showed no statistically significant differences in lipid indices among the different bacterial isolates ($p > 0.05$), indicating that changes in lipid levels are independent of the causative organism.

Table(5): Lipid Profile Parameters According to Bacterial Isolate Type in Group I (n=30)

Parameter (mg/dL)	<i>S.aureus</i> (n=10)	<i>E.coli</i> (n=9)	<i>Streptococcus spp.</i> (n=4)	<i>Klebsiella spp.</i> (n=3)	<i>Pseudomonas spp.</i> (n=2)	P-value
Total Cholesterol	142.40 ± 22.19	157.23 ± 20.89	153.74 ± 25.37	144.66 ± 19.81	137.60 ± 16.59	0.230
Triglycerides	173.44 ± 31.13	157.39 ± 8.73	156.24 ± 34.80	169.40 ± 29.91	177.52 ± 34.14	0.316

HDL-Cholesterol	31.80 ± 6.29	35.19 ± 7.31	34.79 ± 6.90	32.69 ± 6.97	30.56 ± 4.89	0.402
LDL-Cholesterol	79.44 ± 17.77	91.21 ± 18.33	86.50 ± 21.13	81.68 ± 15.35	75.10 ± 11.74	0.117

DISCUSSION:

The results revealed significant alterations in lipid metabolism among these patients—particularly those with bacterial infections thereby broadening our understanding of the complex interplay between inflammation, infection as well as lipid metabolism. Patients with R.A exhibited marked changes in lipid profiles compared to Control group. These changes were most pronounced in patients with bacterial R.A, characterized by a significant decrease in total cholesterol and both (LDL,HDL) , alongside elevated triglyceride levels. These findings align with previous reports documenting lipid metabolic disturbances in inflammatory joint diseases (Myasoedova *et al.*, 2011; Choy *et al.*, 2014). These findings align with previous reports documenting lipid metabolic disturbances in inflammatory joint diseases (Myasoedova *et al.*, 2011; Choy *et al.*, 2014). Inverse relationship between Levels of HDL and inflammatory markers such as CRP ,procalcitonin and ESR supports the hypothesis that inflammation is the primary driver of these lipid alterations, a phenomenon known as "lipid paradox" in rheumatoid arthritis (Myasoedova *et al.*, 2011). These findings align with other Iraqi studies, such as the study by Sulaiman *et al.*, (2023) which demonstrated strong correlations between inflammatory markers& lipids Profile in Iraqi R.A patients and the study by Ali and Abdul-Razzaq (2022), who documented a marked decrease in lipid levels among patients with active disease, noting that these levels returned to normal following treatment with disease modifying antirheumatic drugs.

One of the most significant findings is the greater alteration of lipid levels in patients with bacterial R.A compared to those with non-infectious R.A. These findings are consistent with the acute-phase response to infection, during which lipoprotein metabolism undergoes significant alteration (Feingold and Grünfeld, 2000). During acute bacterial infection, endotoxins-such as LPS stimulate proinflammatory cytokines (interleukin-6, TNF- α and interleukin-1 beta) in turn, inhibit lipoprotein lipase activity and stimulate hepatic triglyceride synthesis, leading to increased blood triglyceride levels. Conversely, cholesterol synthesis is inhibited, resulting in decreased levels both total cholesterol and LDL.(Feingold and Grunfeld, 2000; Lekkou *et al.*, 2014).

It was reported in 2014 that the relationship between lipid indices and procalcitonin a qualitative marker of bacterial infection suggests that the degree of lipid alteration reflects the severity of the infection; this aligns with studies demonstrating the predictive value of lipid changes in patients with septic arthritis (Lekkou *et al.*, 2014).

Synovial fluid analysis revealed highly significant differences between both bacterial and non-bacterial Rheumatoid arthritis groups. Patients with bacterial R.A exhibited a marked increase in white blood cell count consistent with established diagnostic criteria for septic Rheumatoid arthritis (Mathews *et al.*, 2010) along with a marked decrease in glucose levels and an increase in lactate dehydrogenase (LDH) and total protein levels. Strong correlations between blood lipid indices and inflammatory markers in synovial fluid suggest that lipid changes particularly a decrease in HDL-C may serve as indirect indicators of the severity of bacterial R.A.

Analysis of bacterial isolates revealed that *Staphylococcus aureus* (36.7%) and *Escherichia coli* (26.7%) were the most common pathogens, consistent with global epidemiological data (Mathews *et al.*,2010). The relationship between bacterial virulence factors and immune modulation is crucial for understanding changes in lipid levels; bacterial lipopolysaccharides activate the TLR4 receptor and have been implicated in the pathogenesis of R.A(Abdollahi-Roodsaz *et al.*, 2008). The association between lipid disorders and inflammatory markers underscores the importance of assessing cardiovascular risk in R.A, as a chronic inflammatory state can significantly increase such risks; this is supported by the study by Khalil *et al.*, (2026), who investigated APOE genotypes and cardiovascular disease risk in Iraqi R.A patients. Several limitations should be noted, including the relatively small sample size, the cross-sectional design (establishing causal relationships) Potential confounding factors (diet, Physical activity, medications), and the lack of comprehensive molecular characterization of the bacterial isolates. Mechanistic studies exploring the molecular pathways linking bacterial virulence factors, inflammatory mediators, and lipid metabolism will provide valuable insights, as will as role of the microbiome in mediating lipid changes in Rheumatoid arthritis, as highlighted by Ameen *et al.* (2023).

Conclusion: This study shows that patients with Rheumatoid arthritis, especially those with a bacterial infection, exhibit significant lipid dysregulation characterized by decreased levels of total cholesterol, LDL, and HDL, accompanied by increased levels of triglycerides and VLDL. Strong relationship between markers of inflammation and lipid changes as well as *staph.aureus* was the common pathogenic bacteria in septic Rheumatoid arthritis, although lipid profiles did not differ significantly based on the type of bacterial isolate.

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