Correlation Between miRNA-326 Expression and Serum IL-17 Levels in Iraqi Patients with Rheumatoid Arthritis: A Case-Control Study

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Abstract
Background: Rheumatoid arthritis (RA) is a chronic systemic autoimmune condition driven by synovial inflammation and arthritic destruction. MicroRNA-326 (miR-326) regulates Th17 cell differentiation, the major source of IL-17, a key pro-inflammatory cytokine in RA pathogenesis.
Objectives: The study aims to measure the expression level of miRNA-326 and serum IL-17 in RA patients and healthy controls, analyze the correlation between miRNA-326 expressions with that of serum IL 17 levels, as well as evaluate diagnostic value by ROC curve analysis.
Methods: A case-control study (October 2025–February 2026) that include fifty patients who confirmed as RA along with forty healthy subjects ages matched [44,34] from five different hospitals in Najaf/Iraq. The expression of miRNA-326 was evaluated by one-step RT-qPCR (using U6 snRNA as a reference gene and the $2^{-}\Delta\Delta Ct$ method). Sandwich ELISA was used to quantify IL-17. RF was determined by latex agglutination and anti-CCP by qualitative ELISA. Statistical analyses were performed using the Mann–Whitney U test, Spearman's correlation and bootstrap ROC analysis.
Results: miRNA-326 was significantly downregulated in RA patients (mean fold change 0.596 ± 0.387) versus controls (1.265 ± 0.864; $p < 0.0001$). Serum IL-17 was significantly elevated in RA patients (64.89 ± 6.73 ng/L) versus controls (49.64 ± 4.79 ng/L; $p < 0.0001$). A strong negative correlation was observed between miRNA-326 and IL-17 in RA patients (Spearman $r = -0.672$, $p < 0.0001$). ROC analysis yielded AUC values of 0.944 (95% CI: 0.885–0.991) for IL-17 (sensitivity 84.0%, specificity 100.0%; cut-off ≥ 60.50 ng/L) and 0.777 (95% CI: 0.671–0.869) for miRNA-326 (sensitivity 88.0%, specificity 57.5%; cut-off ≤ 1.041). RF positivity was 82% (41/50) and anti-CCP positivity was 94% (47/50).
Conclusion: miRNA-326 is significantly downregulated while IL-17 is markedly elevated in RA, with a strong negative correlation between them. Both biomarkers demonstrate meaningful diagnostic value, supporting the involvement of the miRNA-326/Th17/IL-17 regulatory axis in RA pathogenesis in RA pathogenesis.

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1. Introduction

Rheumatoid arthritis (RA) is an inflammatory autoimmune disease that chronically progress affecting primarily synovial joints. RA is featured by ongoing synovitis, systemic inflammation and autoantibody production which results in joint damage including articular cartilage destruction bone erosion leading to functional incapacitation [1, 2]. RA is estimated to affect ~1% of the worldwide population with a clear female predominance, its incidence further increasing due to global demographic aging [3, 4]. RA is not an immediate cause of death, but the complications related to RA including cardiovascular disease [12], secondary

infections and adverse effects associated with drugs used for treating this pathology are responsible for the excess morbidity that significantly lowers quality of life in these patients [5, 6].

T helper 17 (Th17) cells as well as their signature cytokine interleukin-17A (IL-1725), are among the most important cellular effectors involved in sustaining synovial inflammation and osteoclast-mediated bone destruction during RA pathogenesis [7, 8]. Serum levels of IL-17 are significantly elevated over normal [8], and correlate with validated indices of disease activity in RA, confirming the pathogenetic role for this cytokine as both mechanistic driver and clinically relevant biomarker. Accumulating evidence suggests that epigenetic regulators, especially miRNAs (microRNA), are important mediators in the immunopathogenesis of rheumatoid arthritis by modulating inflammatory signaling pathways and adaptive immune responses. In this way, miRNAs affect T-cell differentiation as well as cytokine production and immune homeostasis thereby participating in the initial steps of disease development but also influencing its progression through post-transcriptional modulation of gene expression.

MicroRNAs (miRNA) are short non-coding RNA molecules (~22 nucleotides), that post-transcriptionally regulate gene expression through base-pairing to the 3' untranslated region of their target mRNAs [10]. Abnormal miRNA expression profiles have been reported in different autoimmune diseases, including RA [11]. MirNA-326, for example, has drawn significant interest due to its role as a regulator of immune cell differentiation. Du *et al*. MiR-326 was shown to directly modulate the 3′UTR of Ets-1 (a transcription factor that inhibits Th17 cell differentiation) and so it could promote IL-17 production by negatively regulating miR-326 [12]. This regulatory axis of miR-326/Ets-1/Th17 has been associated with multiple sclerosis and is proposed to work in a manner analogous for RA [12, 13]. Sui *et al*. miR-326: The expression of miRNA145 is strongly downregulated in the peripheral blood of RA patients 14, and has been shown to have diagnostic AUC = 0.813

These advances notwithstanding, evaluation of miRNA-326 and serum IL17 in tandem with respect to diagnostic performance and association remain sparse despite accumulating evidence for involvement by such elements in the underlying pathogenesis of autoimmune disease as a whole within Middle Eastern populations.

in Middle Eastern populations. This study was conducted to: (i) measure the levels of miRNA-326 in peripheral blood from RA patients and healthy controls recruited from Najaf, Iraq; (ii) Measure serum IL-17 concentrations; (iii) detection if they were correlated with each other as well as iv strain their diagnostic value via ROC curve analysis.

2. Materials and Methods

2.1. Study Design and Setting

This case-control study was carried out from October 2025 to February 2026 at five health care centres in Najaf, Iraq: Al-Sadr Medical City (n = seven patients), Najaf Teaching Hospital (n = twelve)), Al-Zahraa Teaching Hospital (n= ten),Al-Hakeem General Hospital(n=4) and private rheumatology consultations whose physicians were familiar with the complaints of LYVEFvs. Alicia had RA diagnosed by licensed rheumatologists according to the 2010 American College of Rheumatology (ACR)/European League Against Rheumatism (EULAR) classification criteria [15]. Approval from the institutional review board was acquired, and written informed consent was obtained in accordance with the Declaration of Helsinki.

2.2. Participants

Methods: Fifty RA patients (study group) and 40 age- and gender-matched healthy volunteers served as controls. Diagnosis of RA was confirmed based on clinical evaluation, radiography results and positivity for anti-CCP serology and/or RF. Healthy controls were defined as subjects with neither personal nor family history of autoimmune or inflammatory disease and all laboratory parameters within normal reference ranges. Exclusion criteria were: other autoimmune diseases; active malignancy; acute infection and pregnancy as well chronic hepatic or renal insufficiency.

2.3. Blood Collection and RNA Extraction

Venous blood was withdrawn by standard venipuncture (4–5 mL). Blood was separated by centrifugation (2000–3000 rpm, 20 min) to get serum and stored at −80°C; total RNA of peripheral blood retained using the TransZol Up Plus RNA Kit (TransGen Biotech, China). The quality of the RNA was verified by UV spectrophotometry measuring 260/280nm wavenumbers (1.8-2.1).

2.4. miRNA-326 Quantification by RT-qPCR

miRNA-326 expression was quantified using the GoTaq® 1-Step RT-qPCR System (Promega, USA) in 20 µL reactions containing: 10 µL GoTaq® qPCR Master Mix (2×), 2 µL each forward/reverse primer (10×), 0.4 µL GoScript™ RT Mix (50×), 4 µL RNA template, and nuclease-free water to volume. Thermocycler conditions: reverse transcription 37°C/15 min; hot-start activation 95°C/10 min; 40 cycles of denaturation (95°C/10 s), annealing/data collection (60°C/30 s), extension (72°C/30 s); dissociation 60–5°C. U6 snRNA served as the internal reference gene. Relative expression was calculated using the $2^{-}\Delta\Delta Ct$ method [16]. Primer sequences (Table 1) were adopted from Sui *et al*. [14].

Table 1: Primer sequences used for RT-qPCR quantification of miRNA-326 and U6 snRNA.

Gene	Forward Primer (5'→3')	Reverse Primer (5'→3')
miRNA-326	5'-GCAGCACGCTAGGTAGTTTCC-3'	5'-TATCGTTGTTCTCCACTCCTTGAC-3'
U6 (reference gene)	5'-TCCGATCGTGAAGCGTTC-3'	5'-GTGCAGGGTCCGAGGT-3'

Primer sequences adopted from Sui *et al.* (Exp Ther Med, 2020) [14].

2.5. Serum IL-17 Measurement

Serum IL-17A was quantified by sandwich ELISA (BT LAB, China). Each 40 µL serum sample was incubated with biotinylated anti-IL-17A antibody and streptavidin-HRP at 37°C for 60 min, followed by TMB substrate development (10 min, dark, 37°C). Absorbance was read at 450 nm. IL-17 concentrations were calculated from a six-point standard curve (20–320 ng/L).

2.6. RF and Anti-CCP Detection

Rheumatoid factor (RF) was detected by slide latex agglutination (SPINREACT, Spain). Anti-CCP IgG antibodies were measured by qualitative ELISA (BT LAB, China).

2.7. Statistical Analysis

Analyses were performed using IBM SPSS Statistics (version 26) and Python 3 (scikit-learn, SciPy). Between-group comparisons of continuous variables used the Mann–Whitney U test; categorical comparisons used the chi-square test. Spearman's rank correlation coefficient (r_s) quantified the relationship between miRNA-326 and IL-17 in RA patients. ROC curves were made and AUC was calculated; the Youden index (sensitivity + specificity – 1) was used to determine the optimal cut-off point. The 95% confidence intervals (CIs) for AUC values were estimated using the bootstrap resampling method (2,000 resamples; random seed = 42). P value of < 0.05 was deemed statistically significant.

3. Results

3.1. Demographic and Clinical Characteristics

Fifty RA patients (36 females [72.0%] and 14 males [28.0%]; mean age 46.62 ± 11.96 years; range 28–70) and 40 healthy controls (26 females [65.0%] and 14 males [35.0%]; mean age 45.80 ± 10.50 years; range 24–63) were enrolled. No statistically significant differences in age (p = 0.737) or sex distribution were found between groups, confirming adequate matching. Patients were recruited from five facilities: Private Clinic (n = 17, 34%), Najaf Teaching Hospital (n = 12, 24%), Al-Zahraa Teaching Hospital (n = 10, 20%), Al-Sadr Medical City (n = 7, 14%), and Al-Hakeem General Hospital (n = 4, 8%). Demographic and clinical characteristics are summarized in Table 2.

Table 2: Demographic and clinical characteristics of study participants.

Characteristic	RA Patients (n = 50)	Healthy Controls (n = 40)
Age, years (mean ± SD)	46.62 ± 11.96	45.80 ± 10.50
Age range (years)	28 – 70	24 – 63
Female, n (%)	36 (72.0%)	26 (65.0%)
Male, n (%)	14 (28.0%)	14 (35.0%)
Age comparison p-value	0.737 (NS)	

NS: not significant (independent samples t-test).

3.2. Serological Markers (RF and Anti-CCP)

RF was positive in 41 of 50 RA patients (82.0%) and in none of the 40 healthy controls (0%). Of the 9 RF-negative patients, all had RA confirmed by positive anti-CCP (n = 6) or clinical criteria. Anti-CCP antibodies were detected in 47 of 50 RA patients (94.0%); the three anti-CCP-negative patients were also RF-negative, representing seronegative RA. The mean anti-CCP OD in RA patients was 0.700 ± 0.328 (median 0.636; range 0.054–1.398), versus 0.102 ± 0.037 (median 0.097; range 0.050–0.171) in controls, a highly significant difference (Mann–Whitney U = 1950, p < 0.0001). All controls were both RF-negative and anti-CCP-negative, confirming the validity of the control group. Results are presented in Table 3.

Table 3: Serological marker results in RA patients and healthy controls.

Parameter	RA Patients (n = 50)	Controls (n = 40)	p-value
RF Positive, n (%)	41 (82.0%)	0 (0%)	< **0.0001**
RF Negative, n (%)	9 (18.0%)	40 (100%)	
Anti-CCP Positive, n (%)	47 (94.0%)	0 (0%)	< **0.0001**
Anti-CCP Negative, n (%)	3 (6.0%)	40 (100%)	
Anti-CCP OD, mean ± SD	0.700 ± 0.328	0.102 ± 0.037	< **0.0001**
Anti-CCP OD, median (range)	0.636 (0.054–1.398)	0.097 (0.050–0.171)	

RF: rheumatoid factor; OD: optical density; Mann–Whitney U test.

3.3. Serum IL-17 Levels

Serum IL-17 concentrations were significantly higher in RA patients than in healthy controls (Table 4). The mean IL-17 in the RA group was 64.89 ± 6.73 ng/L (median 65.76 ng/L; range 44.94–76.31 ng/L), compared to 49.64 ± 4.79 ng/L (median 49.68 ng/L; range 40.28–60.01 ng/L) in controls. This difference was highly statistically significant (Mann–Whitney U = 1887, $p = 6.09 \times 10^{-13}$; independent t-test: t = 11.956, $p = 3.92 \times 10^{-20}$).

3.4. miRNA-326 Expression

miRNA-326-fold change ($2^{-}\Delta\Delta Ct$) was significantly lower in RA patients than in healthy controls (Table 4). The mean fold change in patients was 0.596 ± 0.387 (median 0.400; range 0.131–1.589), compared to 1.265 ± 0.864 (median 1.082; range 0.184–3.625) in controls, indicating marked downregulation of miRNA-326 in RA. This difference was highly significant (Mann–Whitney U = 446, $p = 7.10 \times 10^{-6}$; t-test: t = −4.843, $p = 5.43 \times 10^{-6}$).

Table 4: Comparison of IL-17 serum levels and miRNA-326 expression between groups.

Parameter	RA Patients (n=50)	Controls (n=40)	U statistic	p-value
IL-17 mean ± SD (ng/L)	64.89 ± 6.73	49.64 ± 4.79	1887	6.09×10^{-13}
IL-17 median (ng/L)	65.76	49.68		
IL-17 range (ng/L)	44.94–76.31	40.28–60.01		
miRNA-326 mean ± SD	0.596 ± 0.387	1.265 ± 0.864	446	7.10×10^{-6}
miRNA-326 median	0.400	1.082		
miRNA-326 range	0.131–1.589	0.184–3.625		

miRNA-326-fold change calculated by $2^{-}\Delta\Delta Ct$ method; Mann–Whitney U test; all p-values < 0.0001.

3.5. Correlation of serum miRNA-326 with IL-17

In the RA patient cohort, Spearman correlation suggested a strong and significant negative correlation between miRNA-326-fold change in serum IL-17 concentration (Spearman $r_s = -0.672$; $p < 0.0001$) as well as Pearson r-values with similar results: −0.712 ± SD 22%; p < 7824934663]]{"type":13,"urlnad} This also demonstrates that RA patients with lower miRNA-326 expression have consistently higher serum IL-17 levels, which directly supports the proposed novel regulatory axis comprising of miRNA-326/Th17(−)/IL-17 (Figure 2).

3.6. ROC Curve Analysis

An analysis of the ROC curve was applied to determine the diagnostic performance for IL-17 and miRNA-326 in differentiating RA patients from healthy controls (Figures 1; Table 5).

AUC for serum IL-17 was 0.944 (95% CI: 0·885–0·991), demonstrating excellent discriminative ability. Using the Youden-optimal cut-off of 60.50 ng/L, IL-17 demonstrated a sensitivity of 84.0% and a specificity of 100.00%, as no healthy control exceeded that when assayed (Fig.unless stated otherwise); J= {28D − [4 C]}, p <. Such uniqueness strongly backs up IL-17 as a very useful confirmation biomarker in RA.

The AUC for miRNA-326 was 0.777 (95% CI: 0.671–0.869) indicating acceptable-to-good diagnostic performance. The optimal cut-off was at ≤1.041-fold change, with a sensitivity of 88.0% and specificity of 57.5%. High sensitivity indicates that miRNA-326 may be a suitable screening biomarker, particularly when combined with conventional serology.

Table 5: ROC curve diagnostic parameters for IL-17 and miRNA-326.

Parameter	IL-17 (ng/L)	miRNA-326 (fold change)
AUC	**0.944**	**0.777**
95% Confidence Interval	0.885 – 0.991	0.671 – 0.869
Optimal Cut-off	≥ 60.50 ng/L	≤ 1.041
Sensitivity	84.0%	88.0%
Specificity	100.0%	57.5%
Youden Index	0.840	0.455

AUC: area under the curve; CI: 95% bootstrap confidence interval.

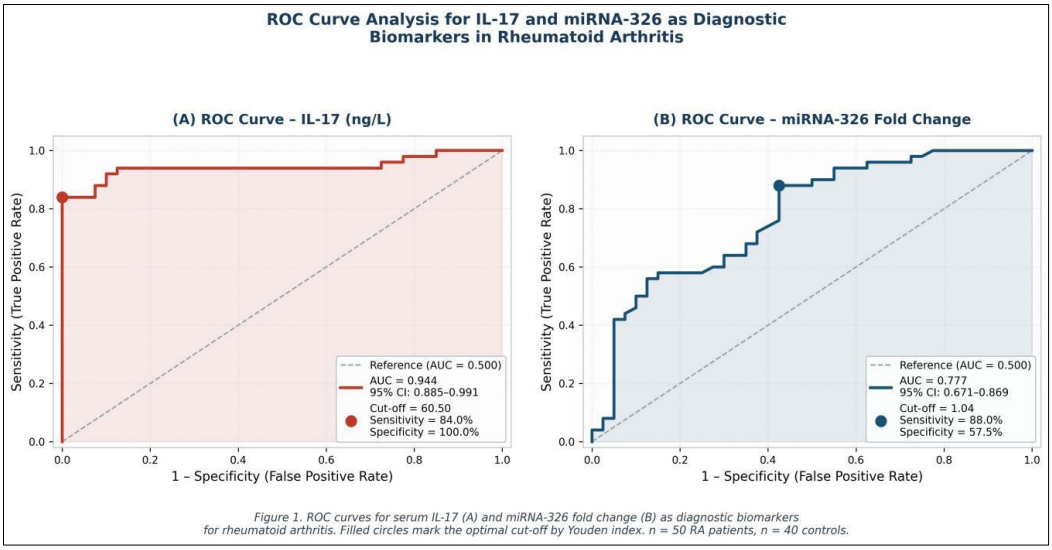

Fig 1: ROC curves for (A) serum IL-17 and (B) miRNA-326 fold change as diagnostic biomarkers for rheumatoid arthritis. Filled circles mark the optimal cut-off (Youden index). AUC: area under the curve; CI: 95% confidence interval. RA patients n = 50; healthy controls n = 40.

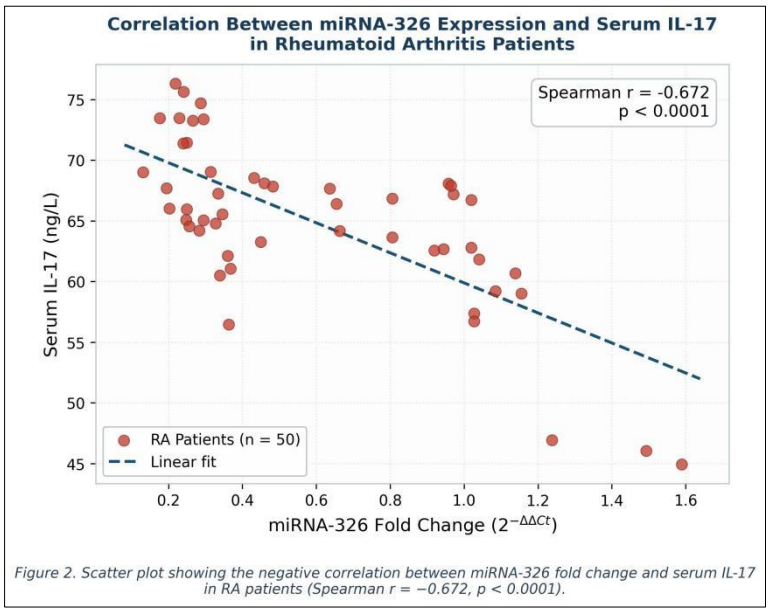

Figure 2. Scatter plot showing the negative correlation between miRNA-326 fold change and serum IL-17 in RA patients (Spearman r = −0.672, p < 0.0001).

Fig 2: Scatter plot showing the strong negative correlation between miRNA-326-fold change ($2^{-}\Delta\Delta Ct$) and serum IL-17 in RA patients (Spearman $r_s = -0.672$, $p < 0.0001$; Pearson $r = -0.712$, $p < 0.0001$; $n = 50$).

4. Discussion

This case-control study adds further evidence for the role of miRNA-326/Th17/IL-17 regulatory axis in pathophysiology of rheumatoid arthritis (RA). The key results showed that serum levels of miRNA-326 in RA patients were significantly lower than normal controls while the concentrations of IL-17 increased notably ($p<0.01$). In addition, these biomarkers were significantly inversely correlated with each other, suggesting concurrent dysregulation of epigenetic regulation and inflammatory signaling as the disease progresses. Taken all together, these results indicate that miRNA-326 and IL-17 give additional information about immune dysregulation in RA as possible biomarkers useful for diagnosing the disease or monitoring it [15, 16].

The marked downregulation of miRNA-326 we report here is consistent with increasing evidence that aberrant microRNA expression plays a major role in dysregulating immunity in disease. MicroRNAs (miRs) are short, non-coding RNA molecules that regulate post-transcriptional gene expression and have been shown to affect T-cell activation, cytokine production and maintenance of immune homeostasis. Molecular diagnostic approaches, including nucleic acid–based technologies, continue to provide increasing evidence of improved understanding about the role(s) played by inflammatory mechanisms across other responsive immune-mediated disorders/reproductive diseases. Thus, decreased expression of miRNA-326 may be an indicator for dysfunctional immunoregulatory mechanisms after which the balance shifts to permit chronic inflammatory responses in RA. Our results agree with previous isolates [14, 17] and are additionally supported by latest critiques of non-coding RNAs as a possible finger for RA diagnostic and pathogenesis together with other information [15, 18].

Previous experimental studies showed that miRNA-326 regulates Th17-cell differentiation by targeting the transcription factors Ets-1, STAT3 and RORγt [12]. The disruption of these signaling networks potentiates IL-17-producing lymphocyte differentiation and amplifies inflammatory responses in the synovium. Though these signaling molecules were not directly measured in the present study, such a decrease in miRNA-326 fits well with proposed models of epigenetic regulation of adaptive immunity for RA [18, 19].

Our patients showed significantly higher serum IL-17 levels which helps to support the key role of Th17 mediated immunity in RA. IL-17 activates fibroblast-like synoviocytes, macrophages, endothelial cells and osteoclasts to produce TNF-α and IL–6 as well as chemokines (such as CCL2) matrix metalloproteinases leading to cartilage degradation bone erosion. Chronic activation of these inflammatory pathways directly drives disease progression and joint destruction, which is reflected in the significantly elevated IL-17 concentrations now observed within this cohort [20, 21].

One of the major baseline findings from this study was that there was a strong inverse correlation between levels outside of miRNA-326 and serum IL17. Lower miRNA-326 expression was associated with consistently higher circulating IL-17 concentrations, indicating that these markers are involved in a shared immunoregulatory pathway rather than independent biological changes. Such reciprocal association thus serves as a form of indirect clinical evidence supporting the conjectured miRNA-326/Ets-1/Th17 signaling axis and further supports that resistance to epigenetic modulation leads to persistent overproduction or excessive inflammatory cytokines contributing RA [16, 20].

Both of the biomarkers were clinically and diagnostically significant. Serum IL-17 possessed good discriminative ability, while miRNA-326 was highly sensitive for identifying affected individuals. Collectively, these results indicate that the use of molecular biomarkers together with conventional serological markers as RF or anti-CCP antibodies may provide additional value in terms of

increasing diagnostic accuracy for early diagnosis and (often) ambiguous cases like those at risk to progress from undifferentiated arthritis towards RA. Strategies based on the use of multiple biomarkers are now being considered a relevant pillar in precision medicine approaches to rheumatology [21, 22].

One of the main strengths of our study is the simultaneous assessment and comparison molecular, immunological, and traditional serological biomarkers in a same cohort of patients. In parallel measurement of miRNA-326 expression, serum concentration IL-17, RF and anti-CCP antibodies; ROC-derived diagnostic performance offers us the comprehensive understanding of epigenetic regulation or inflammatory activities. Additionally, this is one the few studies that consider miRNA-326/IL-17 axis in an Iraqi population thus providing important regional evidence to further corroborate growing international literature.

However, there are some limitations that should be considered. A limitation of our study includes the use a moderate sample size and patients exclusively recruited from 1 geographic region which may limit generalizability. Furthermore, its cross-sectional nature prevents any assessment of temporal changes in either biomarker expression or treatment response. Multicenter longitudinal studies integrating disease activity indices, treatment response and additional signaling molecules (e.g., Ets-1, STAT3 and RORγt) are needed to elucidate the mechanistic/prognostic role of miRNA-326 in RA [23, 24].

In summary, the reciprocal downregulation of miRNA-326 and up-regulated IL-17 along with a robust negative correlation as well as an adequate diagnostic performance indicate that aberrant epigenetic immune regulation plays a molecular basis for persistent inflammation in RA. This insight corroborates the literature on RA immunopathogenesis and will be useful for future applications to develop an integrated molecular biomarker that may lead towards better diagnosis, patient stratification, disease monitoring as well as a target intervention tailored therapeutic [15, 25].

Thus, decreased miRNA-326 expression may indicate an impaired immunoregulatory process that allows for chronic inflammatory response in RA. The interaction between immune-inflammatory processes has been similarly described in other chronic disorders, where metabolic dysregulation, psychological stress and environmental exposures all converge to shape inflammatory signaling as well as the trajectory of disease progression [26].

5. Conclusion

The results show that miRNA-326 is significantly down regulated and serum IL-17 is significantly up-regulated in RA patients from Najaf, Iraq. The significant negative correlation between these molecules (Spearman $r_s = -0.672$, $p < 0.0001$) supports the regulatory relationship between miRNA-326 and Th17/IL-17 directly in the clinical context. ROC analysis yielded IL-17 as a promising biomarker confirmatory biomarker (AUC 0.944; specificity 100%), and miRNA-326 as a sensitive screening marker (AUC 0.777; sensitivity 88.0%) and with complementary diagnostic roles. The results presented provide insight into the immunopathogenesis of RA and suggest that both molecules could be used as a part of a diagnostic panel and as therapeutic targets.

Conflicts of Interest

The authors declare no conflicts of interest.

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Author Contributions

Shlash, A.A.A: Conceptualization, Methodology, Supervision, Validation, Project administration, Formal analysis, Data interpretation, Writing – Original Draft, Writing – Review & Editing.

K.K.A.H.: Investigation, Data Curation, Resources, Laboratory Investigation, Formal analysis, Visualization, Writing – Original Draft, Writing – Review & Editing.

All authors contributed to the interpretation of the findings, critically revised the manuscript for important intellectual content, approved the final version of the manuscript, and agreed to be accountable for all aspects of the work.

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