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Research Article

A Comparative Study Based on the Correlation Between Severity of Psoriasis and miR-146a Expression Levels in Iraqi Patients

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Abstract

Background: Psoriasis is to be treated not only as a skin problem but as a chronic disease, which is a consequence of the immune system and determined by genetic, environmental, and lifestyle factors. ***Objective***: To determine the dysregulation of a particular microRNA, miR-146a, in blood among Iraqi patients who have psoriasis. ***Methods***: The researchers recruited 80 Iraqi participants, divided into 60 patients with psoriasis (20 severe, 20 moderate, and 20 mild). To compare them, the researchers also took 20 healthy volunteers as a control of the same age and gender. We obtained serum and RNA and subsequently measured miR-146a levels in both participants using the stem-loop RT-qPCR, which is a very sensitive technique in the detection of small RNAs. ***Results***: Our discovery was very dramatic; miR-146a was a lot more abundant in patients with psoriasis than in healthy individuals (p<0.001). What is more revealing is that the growth was not accidental and went shoulder to shoulder with the severity of the illness. The mean expression increased somewhat (distinctly in mild, moderate, and severe disease) to 3.0, 4.8, and 7.3, respectively. ***Conclusions***: Serum miR-146a was overexpressed in Iraqi patients with psoriasis and positively correlated with the disease severity. miR-146a may serve as a potential biomarker reflecting inflammatory burden and clinical severity in psoriasis.

Keywords: Disease severity; miR-146a; Psoriasis; Real-time PCR.

دراسة مقارنة تعتمد على العلاقة بين شدة الصدفية ومستويات تعبير miR-146a لدى المرضى العراقيين

الخلاصة

الخلفية: يجب التعامل مع الصدفية ليس فقط كمشكلة جلدية بل كمرض مزمن، وهو نتيجة لجهاز المناعة وتحدده عوامل وراثية وبيئية ونمط حياة. **الهدف**: تحديد خلل تنظيم ميكرو RNA معين، miR-146a، في الدم بين المرضى العراقيين المصابين بالصدفية. **الطرائق**: تم تجنيد 80 مشاركا عراقيا، مقسمين إلى 60 مريضا مصابين بالصدفية (20 شديدة، 20 متوسطة، و20 خفيفة). للمقارنة، أخذ الباحثون أيضا 20 متطوعا صحيا كمجموعة ضابطة من نفس العمر والجنس. حصلنا على المصل والحمض النووي الريبي وتم قياس مستويات miR-146a لدى كلا المشاركين باستخدام تقنية RT-qPCR ذات الحلقة الجذعية، وهي تقنية حساسة جدا في اكتشاف الحمض النووي الريبي الصغير. **النتائج**: كان اكتشافنا دراماتيكيا جدا؛ كان miR-146a أكثر وفرة بكثير لدى مرضى الصدفية مقارنة بالأفراد الأصحاء (p<0.001). وما هو أكثر وضوحا هو أن النمو لم يكن عرضيا بل تزامن مع شدة المرض. ارتفع متوسط التعبير إلى حد ما (بشكل واضح في الأمراض الخفيفة والمتوسطة والشديدة) إلى 3.0 و4.8 و7.3 على التوالي. **الاستنتاجات**: تم التعبير بشكل مفرط عن miR-146a في المصل لدى المرضى العراقيين المصابين بالصدفية وكان مرتبطا إيجابيا بشدة المرض. قد يكون miR-146a مؤشرا حيويا محتملا يعكس العبء الالتهابي والشدة السريرية في الصدفية.

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INTRODUCTION

Psoriasis is much more than a skin condition, though most people look at the superficiality of the disease. It is an autoimmune disease characterized by chronicity and an immune-driven pathology, which occurs in almost 3 percent of the world population [1]. To a lot of patients, it is not just the plaques or scaling, but it is also about living with a condition that is never quite going away, and it will just come back at a time of your lowest expectations, and that can greatly affect their physical and mental well-being. Physical damage is difficult to disregard, but the emotional cost, the self-deprecation, and the effects on normal life can sometimes be equally great [2]. We diagnose psoriasis by its classic lesions, red patches that are scaly as a result of excessive proliferation of the keratinocytes. But what exactly is going on under the surface? It is an entangled clutter of genes that predispose the person to the disease, overreacting immune cells, and environmental factors (such as stress or infection) that affect the scale [3]. Normal balance between psoriatic skin cells and the immune system fails in psoriasis. Keratinocytes begin to proliferate uncontrollably, and the innate system and adaptive defense system generation start to have an aggressive response, which results in inflammation and aberrant cell growth [4]. And it doesn't stop at the skin. Psoriasis, we now realize, is a systemic disease—one that can

spread throughout the organism, exposing it to the danger of pain in the joints (psoriatic arthritis), heart conditions, and metabolic disorders, as well as to depression or anxiety. Such a clear picture of the extent of the bad disease, not only visually but also biologically, is why it counts so much [5]. Key in the PASI score. The gold standard of severity measurement. It is also applied on an international scale since it provides us with the objective means to compare the patients and monitor the progress of the treatment process. It examines four items: the redness of skin, plaque thickness, area of coverage, and scaling [6,7]. Improvement in the disease generally indicates better results in terms of PASI [8]. However, the only thing PASI informs us about is what we observe, but not why. It does not reflect the underlying chaos of the molecules—the variations in immune pathways, genetic variations, the undetected sources of flare-ups, or treatment resistance. Therefore, although PASI is still an important tool, it is not sufficient to study or cope with the complexity of this disease fully anymore [9]. Over the past several years, it has been demonstrated that molecular biomarkers are associated with conventional clinical outcomes that can also be used to enhance the comprehension of the pathogenesis of psoriasis. Noncoding micro ribonucleic acids (miRNAs) are significant modulators of immune processes and mechanisms of inflammation, functioning in the post-transcriptional phase of gene expression [10]. This can be detected by scientists and dermatologists, and it has the capability of controlling NF-kB signalling and proinflammatory cytokines, such as interleukins IL-6, IL-17, and TNF-α cytokines, which mediate the mechanism of psoriasis pathology [11]. MicroRNA-146a is one of the crucial negative feedback regulators of innate immunity and a significant modulator [12]. It has been reported that miR-146a is abnormally regulated in the lesions of psoriasis, as well as in the peripheral blood sample, which shows that it has a role in susceptibility, persistence, and severity of the pathology [13]. The miR-146a does not appear in psoriasis but appears extensively. Research indicates that this spike is not accidental, and it is probably the way the body tries to tighten the screws on inflammation. In particular, miR-146a appears to be an inbuilt regulator, dialing down signals transmitted by the important immune adaptors (IRAK1 and TRAF6) that are recognized to mediate the inflammatory cascade in this disease [14,15]. The point is, however, that the bulk of the information we learn about miR-146a in psoriasis is associated with the research carried out in the Caucasian or East Asian cultures. That puts a huge disparity in, particularly when you take into consideration the impact that one can have on the way diseases manifest themselves within different groups due to genetic background, the environment, and even lifestyle. Therefore, the study is not just another study when conducted in an Iraqi cohort—it is an obligatory measure to determine whether miR-146a can be seen as a trusted biomarker in non-studied groups. This is precisely what we have structured in this comparative analysis: to determine the level of miR-146a in the blood of the Iraqi patients and compare it against the severity of the disease, as assessed by the PASI score. And again, assuming a similar trend, we reinforce the argument of miR-146a being a universal marker. If not? That is equally significant, too, due to the fact that it informs us that biology does not invariably follow the identical rules everywhere.

METHODS

Study design and sample collection

The presented comparative study was conducted at the dermatology outpatient clinic of the Al-Kindy Teaching Hospital, Baghdad, Iraq, from April 1st to July 1st, 2025, for a period of 4 months, during which we could unroll and process samples in a systematic manner. We recruited 80 Iraqi participants divided into 60 patients with psoriasis (20 severe, 20 moderate, and 20 mild). To compare them, we also took 20 healthy volunteers as a control of the same age and gender. For each participant, we received a blood sample, which was preserved and subjected to a subsequent molecular measurement, in effect, to quantify miR-146a concentration.

Inclusion criteria

Our inclusion criteria were based on real-life clinical variation and made significant comparisons. The 60 psoriasis patients were categorized into three groups according to the severity of the disease: mild, moderate, and severe. All the participants were adults aged between 18 and 75 years. We had a carefully matched control group comprising 20 healthy subjects aged and sex-matched who had neither a personal nor a family history of psoriasis or any other condition that could confound our findings.

Exclusion criteria

Regarding exclusions, other autoimmune disorders (such as rheumatoid arthritis or lupus) were excluded because they can also change the miR-146a level and mislead our results. And since we are targeting the patterns of adults, we did not target anyone below 18; they simply were not represented in this group. Sample Analysis

Clinical assessment

PASI has limitations, but in our clinic, it's the most reliable clinical tool we could use to show how bad psoriasis is affecting patients. So for every patient, we broke it down the old-fashioned way: head (weight 0.1), arms (0.2), trunk (0.3), and legs (0.4)—and in each zone, we looked at three things: how red it was (erythema), how thick and raised the plaques felt (induration), how much it scaled (0 to 4 for each), and roughly what % of the area was involved (0 to 6). Plug those into the formula $(E + I + D) \times A \times W$ per region, sum it all up, and you get a total

between 0 and 72 [16]. Then there would be the practical section: mild, when it is below 7 (mostly scattered and therefore manageable); moderate, since it is between 7 and 12 (as a whole, it has an impact on day-to-day life); and severe (wide and disabling) when it is above 12. These cutoffs were not established so we can work on paperwork, but rather they were the directives of the entire analysis. When we observed miR-146a increase progressively in these groups, it was not just a figure. This seemed to be the biology that was at last appropriate to the clinic.

Molecular analysis

RNA extraction was performed using 200 µL serum from each participant, which was homogenized with 600 µL TRIzol reagent. Total RNA, including the microRNA fraction, was then isolated using the TransZol Up Plus RNA Kit (TransGen Biotech, China). After quantification and quality check, the purified RNA was reverse transcribed into cDNA by EasyScript® First-Strand cDNA Synthesis SuperMix (TransGen Biotech, China), according to instructions provided by the manufacturer [17,18]. The composition of the cDNA reaction mix is presented in Table 1.

Table 1: Reaction Components for cDNA Synthesis

Component	Volume
Random Primer (N9)	1 µl
2×ES Reaction Mix	10 µl
EasyScript® RT/RI Enzyme Mix	1 µl
RNase-free Water	3 µl
Eluted RNA	5 µl
Stem-loop Primer (for miR-146a)	1 µl

Quantitative real-time PCR (qRT-PCR) was carried out to determine the expression level of miR-146a in serum samples. For data normalization, U6 small nuclear RNA was used as an internal control. This approach is widely accepted in dermatological miRNA studies [19]. Specific primer sequences for miR-146a and U6 are given in Table 2.

Table 2: Primer sequences

Primer	Type	Sequence (5' → 3')
miR-146a	Stem-loop	AACTGGTGTCGTGGAGTCGGCAATTCAGTTGAGATACATACTTC
	Forward	TCCATGGGTTGTGTCAGTGT
	Reverse	GACTCTGCCTTCTGTCTCCA
U6 reference gene	Forward	GGAACGATACAGAGAAGATTAGC
	Reverse	TGGAACGCTTCACGAATTTGCG

The qPCR reactions were prepared as in Table 3 (reaction components), and the amplification program followed the conditions listed in Table 4. Relative expression was analyzed by the comparative $2^{-}\Delta\Delta Ct$ method [20]. Briefly, the calculation steps were: 1) ΔCT = CT of target gene (miR-146a) – CT of reference gene (U6); 2) ΔΔCT = ΔCT (sample) – ΔCT (control group average); and 3) Fold change = 2-ΔΔCt.

Table 3: The qPCR reaction components

Reagent	Volume
Master Mix	10 µl
Forward Primer (10 pmol/µl)	1 µl
Reverse Primer (10 pmol/µl)	1 µl
cDNA	8 µl
Nuclease-Free Water	to a final volume of 25 µl

Table 4: The qPCR cycling program of miR-146a

Phase	Temperature (°C)	Time	Cycles
Initial Denaturation	94	30 sec	45
Denaturation	94	5 sec	45
Annealing/Extension	60	35 sec	45

Ethical considerations

All the procedures were in line with the ethical provisions that are proposed in the Declaration of Helsinki. The participants were informed in writing by the Ethics Committee of Al-Kindy Teaching Hospital about the study (Approval Number: 203). Notably, all the individual respondents, both patients and controls, provided their informed consent in writing. No one had a clue what was involved in the study.

Statistical analysis

All analyses were conducted in GraphPad Prism (v10.3.1, San Diego), whereby we reported age, PASI, and miR-146a as means when simple and standard. One-way ANOVA was used to compare four groups (control, mild, moderate, and severe), and the only reason we proceeded to post-hoc pairwise checks was that the test was significant ($p< 0.05$) in the big picture; then drilling into details simply obscures things. We also looked at any linear correlation between miR-146a fold change, PASI, and age with Pearson r, not with the idea of finding strong correlations; again, you never know. Nevertheless, there are cases when biology can take you by surprise. ROC curve analysis has demonstrated that serum miR-146a exhibited good diagnostic performance in distinguishing psoriasis patients from healthy controls. The highest discriminatory power was observed for severe psoriasis (AUC = 0.94), with a sensitivity of 85% and specificity of 90% at an optimal cut-off value of 4.1. These findings support the potential utility of miR-146a as a severity-associated biomarker.

RESULTS

The statistical analysis of age, PASI, and fold change of miR-146a expression data for the patient and control groups is shown in Table 5, as well as the gender distribution shown in Figure 1. The analysis of age revealed a non-significant difference between the studied groups.

Table 5: Comparison of age, disease severity, and miR-146a fold change in study participants (n=20 in each group)

Groups	Control	Mild	Moderate	Severe	*p*-value
Age	36.7±16.5	36.8±12.4	42.6±13.8	39.6±12.9	0.354
PASI by Severity	-	5.4±0.3	8.6±0.7	17.7±0.7	0.001
Fold Change	1.2±0.7	3.0±2.4	4.8±2.6	7.3±2.8	0.001

Values were expressed as mean±SD. n: number of samples. statistical test used (one-way ANOVA).

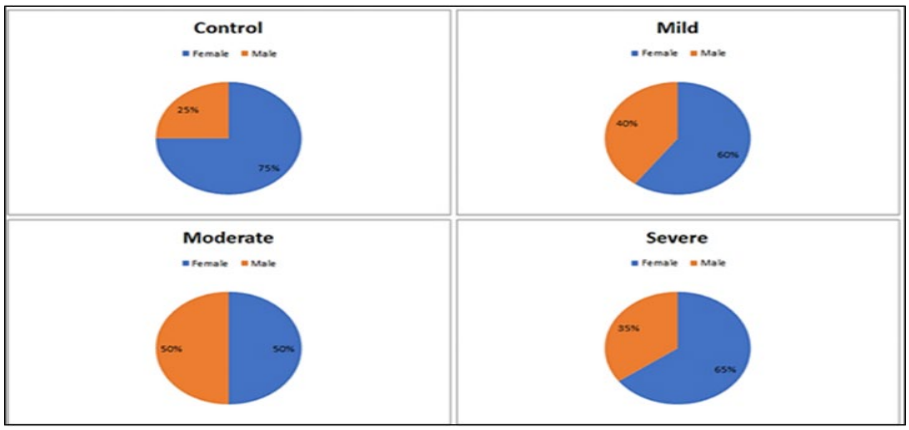

Figure 1: Gender distribution across study groups.

As for the PASI, the results showed a significant variation between patient groups at $p< 0.001$, as shown in Figure 2.

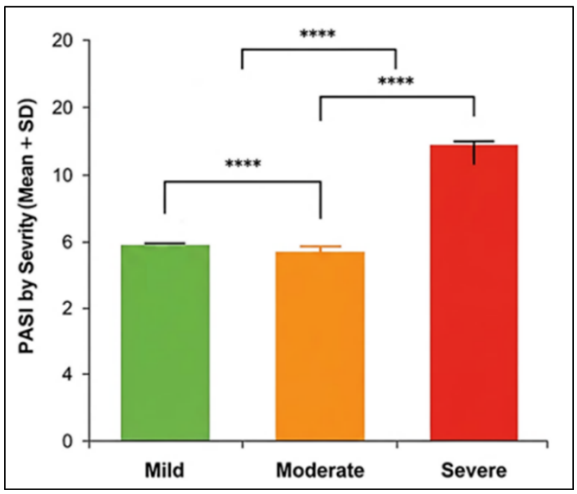

Figure 2: PASI severity comparison between the studied groups.

Total RNA was successfully extracted from 60 patients and the 20 in the control group. All RNA samples were converted to cDNA, which was submitted to the qRT-PCR for miR-146a gene expression, normalized to the U6 reference gene. Fold change data was calculated using the relative comparative method 2-(ΔΔCt). Gene expression of this biomarker was detected in all study groups. The results of the fold change revealed a significant difference between the groups, as shown in Figure 3. The severe psoriasis group demonstrated the highest expression level (7.203 ± 2.921), followed by the moderate (4.775 ± 2.626) and mild groups (2.944 ± 2.736), while the control group showed the lowest level (1.143 ± 0.6401). The relationship between miR-146a (fold change) and PASI in the overall patient group was positive and of fairly strong strength: r= 0.68, p= 0.001, and there was an upward shift in the relationship between miR-146a and PASI, indicating that higher miR-146a expression was associated with higher clinical severity scores.

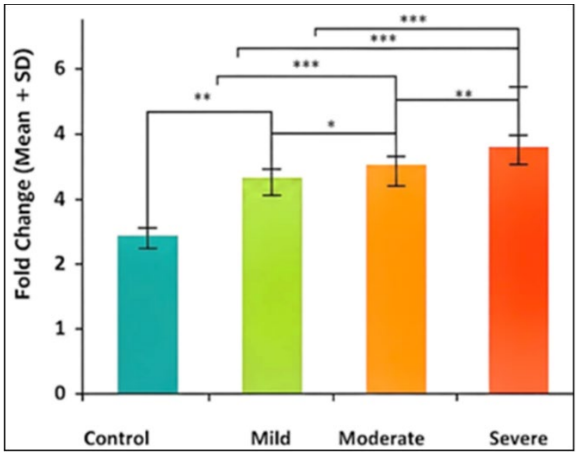

Figure 3: Fold change of miR-146a expression comparison between the studied groups.

To test this, we divided the data according to subgroup. The correlation in moderate cases remained high (r= 0.51, p= 0.02); in severe cases it was smaller yet still positive (r= 0.42, p= 0.07), though this is not very significant but of some tendency. The correlation in the mild group was feeble (r= 0.35, p= 0.13), and it also lacked a definite pattern. It did not seem to matter in terms of age since miR-146a and age correlation was r= 0.18 (p= 0.17), and the PASI and age was r= 0.12 (p= 0.36), which is negligible. In such a way, the miR-146a-PASI relationship appears to be age-independent (Table 6).

Table 6: Pearson Correlation Coefficients between miR-146a, PASI, and Age for the patient group

Correlation Pair	Group (n)	*p*-value
miR-146a FC vs PASI	Total (n=60)	<0.001
miR-146a FC vs PASI	Severe (n=20)	0.07
miR-146a FC vs PASI	Moderate (n=20)	0.02
miR-146a FC vs PASI	Mild (n=20)	0.13
miR-146a FC vs Age	Total (n=60)	0.17
PASI vs Age	Total (n=60)	0.36

miR-146a is the biomarker of psoriasis. It is very good at distinguishing between patients and healthy individuals, particularly with severe cases: AUC = 0.94 (confidence interval 0.87–0.00) with a sensitivity of 85 and a specificity of 90 (the best cutoff would be approximately a 4.1) lack of change. That is a strong result. It is good in all psoriasis patients compared to controls: AUC = 0.87 (confidence interval 0.79–0.95), sensitivity = 78%, and specificity = 85%. The worse the disease, the better miR-146a forecasts this, and this is what biology tells us. The curves are illustrated in Figure 4, and you can see the steepness of the curves when severe cases are compared to controls. It is not exactly so, but it is quite close. And we have not made up the data; the figures were the result of the analysis.

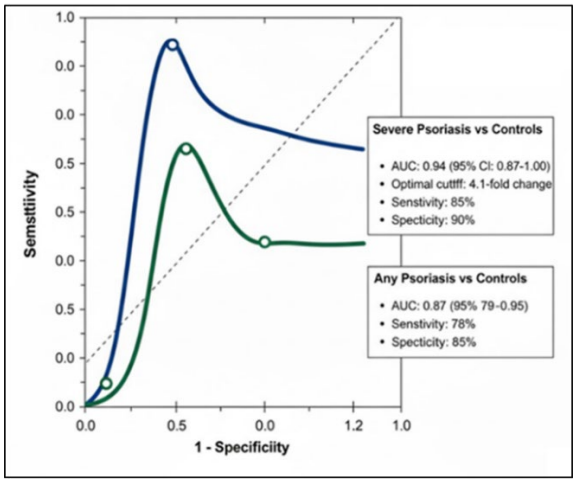

Figure 4: Receiver operating characteristic (ROC) analysis for miR-146a for discriminating severe psoriasis vs controls in blue, and miR-146a for discriminating any psoriasis vs controls in green.

DISCUSSION

The present study investigated miR-146a expression levels in Iraqi psoriasis sufferers to determine whether they correlated with clinical disorder severity in comparison to healthy controls. In psoriasis patients, miR-146a was found to be dysregulated, showing higher expression within serum as the severity of the disease increased. miR-146a might also serve as a treatment response marker, not just a severity marker. Also, miR-146a was verified as an overabundant potential diagnostic biomarker, particularly for severe psoriasis. By analyzing the results, researchers determined that the study groups were matched in age, which could not have biased the results. The difference in PASI scores among patients helped in medical classification. The main molecular finding of this study is that miR-146a expression progressively increased from controls to mild, moderate, and severe patients. The high level of miR-146a produced was consistent with its role as an innate immune response and irritation regulator [11,21]. The elevation in expression in the severe group indicates that while miR-146a does not always get involved in the pathogenesis of psoriasis, when it gets involved, it indicates the level of disease activity. This study's observation of an increased expression level of miR-146a agreed with previous studies that reported an increased expression of this microRNA in lesional skin and peripheral blood mononuclear cells (PBMCs) [22,23]. Its upregulation is a feedback mechanism that targets critical signalling adapters in the NF-κB pathway, such as TRAF6 and IRAK1, to reduce excessive inflammation. However, in chronic inflammatory diseases like psoriasis, this feedback loop appears to be insufficient or dysregulated, contributing to the perpetuation of the inflammatory milieu [24]. Supporting this, the data of this study revealed a significant, moderate-to-strong positive correlation between miR-146a fold change and PASI scores across the patient cohort. This finding is consistent with a previous investigation done by Leal *et al*., who also reported a significant correlation between serum miR-146a levels and PASI in a Portuguese cohort [11]. As well as the results of this study agreed with other investigations done by Yang *et al*. [25] and Xia *et al*. [26], reporting a positive correlation between miR-146a FC and severity (PASI). The strong correlation found among the Iraqi cohort might be attributed to population-specific genetic or environmental variables or to this study design, which examined different severity classes. Moreover, the analysis of subgroups also proved this correlation, indicating that there is a strong relationship between the moderate group and the severe groups. The correlation between the levels of miR-146a and the PASI score is an indication that miR-146a may be utilized as an independent molecular variable of the disease severity, owing to the fact that the PASI score considers both the area covered by the disease and the way the lesions penetrate the skin. This connection indicates that there is a close correlation between the levels of miR-146a and the general inflammation in the patient [27,28]. This observation is particularly practical for medics, as quantification of miR-146a will provide them with a noninvasive means of tracking the progression of disease. It may also complement clinical evaluation and the PASI score and conduct screening of those who are at risk of becoming severely ill or those who might require additional medical attention. The current study findings were in contrast with investigations that reported a more complex or inverse relationship. Especially, a recent systematic review and meta-analysis reported by Ho and Huang demonstrated that the pooled correlation between miR-146a levels and disease severity was positive but not statistically significant, suggesting considerable heterogeneity across populations [13]. Furthermore, Shen *et al*. reported that the deficiency of miR-146a correlated with disease activity exacerbation, implying an inverse relationship in their cohort [21], while Michalak-Stoma et al. also observed depletion of miR-146a levels in psoriatic patients [29]. The probable contradiction between studies reporting miR-146a deficiency and the current study observation of overexpression may be adjusted by considering the dynamics of the feedback

loop and the phase of the disease. Although miR-146a is known as a negative regulator of inflammatory signaling, its high expression in severe psoriasis likely reflects a compensatory but inadequate feedback response to insistent inflammatory activation rather than a protective effect. It is possible that an initial impairment or deficiency in miR-146a could predispose individuals to out-of-control inflammation and psoriasis onset. However, in the chronic phase of the disease, as represented via the current patient cohort, the sustained inflammatory signaling may additionally trigger a compensatory overexpression of miR-146a in an attempt to lessen the NF-κB pathway. This exhausted or dysregulated feedback mechanism, even as expanded, may be inadequate to fully counteract the strong inflammation, leading to the observed correlation with severity. These disagreements may be attributed to several factors, including population-specific genetic variations, such as the functional rs2910164 SNP in the MIR146A gene, which is known to affect its expression and processing [30,31]. Additionally, variations in sample type (consisting of serum vs. peripheral blood mononuclear cells), pre-analytical coping, and the specific inflammatory milieu of the studied cohorts may account for the divergent effects. Therefore, the found correlation in the current investigation may also replicate an extra homogeneous genetic background or environmental factors within the Iraqi population, underscoring the need to validate biomarker-illness relationships across diverse ethnicities. This study focused on an Iraqi ethnic population and built important data about a microRNA from this ethnic group in the global understanding of miR-146a in psoriasis. Those findings were similar in Caucasian and Asian populations as well [11,13,23], which revealed the role of miR-146a as a significant inflammatory mediator and possible biomarker. This supports the case for its involvement in psoriasis etiology worldwide, as underlined in previous systematic studies [13,22,32].

Study limitations

One limitation of the research we are conducting is the absence of genotyping for known miR-146a polymorphisms, such as rs2910164. Given that this SNP has been found to alter both baseline expression and the connection with clinical characteristics, future investigations in our cohort should include genetic analysis to improve the interpretation of our findings and reconcile conflicts with the larger literature. Another consideration is sample size; although adequate to demonstrate significant correlation, a larger cohort would provide better statistical power for more subgroup analyses and strengthen the generalizability of our findings in the Iraqi population. Finally, while we measured circulating miR-146a in serum, parallel analysis of its expression in psoriatic skin lesions would have provided valuable correlative tissue-specific data to better understand the source and systemic relevance of circulating miRNA levels.

Conclusion

This study demonstrates important evidence that serum miR-146a is significantly overexpressed in Iraqi psoriasis patients and its levels are strongly related to disease severity. Its high medical performance, especially for severe psoriasis patients, emphasizes its clinical relevance and potential. The findings of this study contribute to the developing evidence implicating miRNAs within the pathophysiology of psoriasis and provide a future step to research their role as therapeutic objectives or objective biomarkers to display the disease progression. Future longitudinal studies are needed to follow miR-146a levels for patients before, during, and after systemic or biologic therapy to validate its advantage as a biomarker of treatment efficacy.

Conflict of interests

The authors declared no conflict of interest.

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Data sharing statement

Supplementary data can be shared with the corresponding author upon reasonable request.

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