ORIGINAL ARTICLE

Impact of Treatment on Clinicopsychological Status of Rheumatoid Arthritis Patient: A Prospective Observational Study from Tertiary Care Center in Central India

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Received: 18 September 2025; *Accepted:* 12 January 2026

Abstract

Background: Rheumatoid arthritis (RA) is an autoimmune disorder of chronic nature, presenting with inflamed joints and widespread systemic manifestations. Psychological comorbidities such as depression and anxiety are common but often under-recognized, contributing to poor treatment outcomes. The objective of this study is to evaluate the prevalence of depression and anxiety in newly diagnosed RA patients and assess the impact of disease-modifying antirheumatic drug (DMARD) therapy on clinical and psychological outcomes.

Materials and methods: This prospective observational study included 150 treatment-naïve RA patients diagnosed using ACR/EULAR criteria. After applying exclusion criteria and accounting for follow-up losses, 116 patients were included in the analysis. Baseline disease activity was measured using DAS28, while psychological status was assessed using the Hamilton Depression Rating Scale (HDRS) and Hamilton Anxiety Rating Scale (HAMA). Patients received standard DMARD therapy and were reassessed after 6 months.

Results: Among 116 patients, 65.3% had depression and 57.3% anxiety at baseline. Mean scores reflected high disease activity and psychological distress (DAS28: 5.28 ± 1.06, HDRS: 15.9 ± 8.4, HAMA: 11.7 ± 6.8). After 6 months of DMARD therapy, all showed significant improvement: DAS28 (4.60 ± 1.25; $p < 0.001$, $d = 0.53$), HDRS (11.3 ± 6.4; $p < 0.001$, $d = 0.55$), and HAMA (9.0 ± 5.4; $p < 0.001$, $d = 0.37$). The proportion without depression nearly doubled (16.5%→31.2%), with severe depression falling from 23.9% to 5.5% (OR = 7.79, 95% CI 2.92–20.76). Anxiety scores also showed a favorable trend, with the proportion without anxiety increasing from 27.5% to 40.4% and severe cases reducing from 7.3% to 1.8% (OR = 2.83, 95% CI 1.12–7.15).

Conclusion: DMARD therapy not only improves disease activity in RA but also reduces psychological distress. Routine mental health screening and integrated management are essential to optimize patient outcomes and quality of life.

Journal of The Association of Physicians of India (2026): 10.59556/japi.74.1476

Introduction

Rheumatoid arthritis (RA) is an autoimmune disorder of chronic nature, presenting with inflamed joints and widespread systemic manifestations. The continuous inflammatory response in RA not only impairs joint function but also severely affects physical, psychological, and social domains of patient well-being. Although RA predominantly affects synovial joints, extra-articular involvement frequently occurs, significantly reducing patients' quality of life (QoL) and functional capacities.[1,2] The global prevalence of RA is approximately 0.5–1%, with a notably higher prevalence observed in women; its onset commonly peaks during middle age. In India, epidemiological data suggest RA prevalence rates between 0.3% and 0.75%, indicating substantial public health implications within the region.[3,4]

Clinically, RA typically presents with symmetrical joint pain, stiffness, swelling, and eventual deformities, primarily affecting the small joints of the hands and feet, although larger joints may also become involved as the disease progresses. Diagnostic evaluation often relies on serological markers such as anticitrullinated protein antibodies (ACPA) and rheumatoid factor (RF), both of which are critical in identifying and managing seropositive RA cases.[5] Beyond musculoskeletal involvement, RA is associated with various systemic complications, including dermatologic, pulmonary, cardiovascular, and ocular manifestations, collectively contributing to reduced life expectancy—estimated at approximately 5–10 years shorter compared to the general population.[6] Thus, comprehensive and multidisciplinary management addressing both articular and extra-articular manifestations is crucial for optimal patient outcomes.[2,3]

Beyond its physical effects, rheumatoid arthritis greatly affects the mental well-being of patients. The chronicity and unpredictability of symptoms—particularly persistent pain, fatigue, and functional limitations—result in considerable emotional distress, often manifesting as anxiety and depression.[7] Psychological morbidity in RA patients is exacerbated by functional disabilities that compromise the ability to perform daily tasks, social participation, employment, and family roles, thereby deepening the psychosocial burden.[8] Accurately assessing RA severity not only involves monitoring disease activity but also understanding the intricate relationship between physical manifestations and psychosocial factors influencing patient perceptions of their illness.[9]

Despite therapeutic advances, including disease-modifying antirheumatic drugs (DMARDs) and biologic agents aimed primarily at reducing inflammation and disease progression, the psychological dimensions of RA management frequently remain overlooked in clinical practice. Among psychiatric comorbidities, depression represents a substantial burden, with reported prevalence rates ranging widely from 14% to 48% among RA populations.[10] This variability reflects differences in patient populations, assessment tools, and study methodologies. The psychological burden of RA arises not only from chronic disease-associated stressors, such as pain and disability, but also from medication side effects and the psychosocial implications of chronic illness, further contributing to depressive symptoms.[11]

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How to cite this article: Gupta K, Pandey VP, Dubey S, *et al.* Impact of Treatment on Clinicopsychological Status of Rheumatoid Arthritis Patient: A Prospective Observational Study from Tertiary Care Center in Central India. J Assoc Physicians India 2026;74(5):e11–e15.

Various studies globally have documented diverse prevalence rates of anxiety and depression among RA patients, further complicating a comprehensive understanding of psychiatric morbidity in RA.[12,13] Depression prevalence rates have been reported to range widely from 6% to as high as 66%, while anxiety rates have varied even more considerably, from approximately 2.5–70%.[14,15] Methodological variations, such as differing diagnostic criteria or tools [e.g., the Hospital Anxiety and Depression Scale (HADS) versus ICD-10-based criteria], account for significant differences across these studies.[12,13] Studies utilizing HADS typically report lower prevalence rates for depression and anxiety, whereas higher rates are found when diagnostic evaluations are more comprehensive.[14–16] Within the Indian context, a study employing HADS found anxiety and depression rates of 25% and 17%, respectively—substantially higher than general population rates in India, reported between 2.6% and 15.1%.[17,18]

The presence of anxiety and depression significantly impacts patients' subjective experiences and objective clinical outcomes in RA. Patients suffering from psychological comorbidities typically report higher Disease Activity Scores (DAS) due to increased pain sensitivity, amplified reports of joint tenderness, and poorer patient-reported global assessments—even in scenarios of controlled inflammation or clinically inactive disease.[19]

Higher depression scores specifically correlate with increased pain intensity, reduced adherence to prescribed treatments, poorer prognosis, and significantly impaired QoL, establishing depression as an independent risk factor for adverse RA outcomes.[20] The interaction between psychological and physical dimensions of RA underscores the necessity of integrating mental health considerations into routine RA management strategies to enhance overall therapeutic effectiveness.[12–15]

Recent advances in understanding the neuroimmune pathways underlying depression suggest a close interplay between immune-mediated inflammation and psychiatric conditions, proposing a potential shared pathogenic mechanism with RA. Elevated systemic inflammation markers often observed in RA have been increasingly implicated in the pathophysiology of depression, further highlighting the necessity of holistic approaches addressing both conditions concurrently.[19]

Current RA management strategies emphasize controlling disease activity through pharmacological interventions, predominantly using DMARDs and biologics. Although these medications effectively manage inflammation and joint damage, psychological morbidities demand additional interventions such as cognitive-behavioral therapy (CBT), psychosocial support, stress reduction strategies, and structured patient education. Such adjunctive approaches have demonstrated notable improvements in patients' coping mechanisms, psychological health, and QoL.[20,21]

Despite advances in RA treatment and management guidelines advocating "treat-to-target" and "tight control" approaches, clinical remission rates remain suboptimal, partly due to under-recognized and inadequately addressed psychological comorbidities.[22] This situation is particularly pronounced in developing countries such as India, where research on the psychological aspects of RA remains limited. Consequently, a knowledge gap persists regarding the interplay between treatment regimens, clinical outcomes, and psychological well-being within Indian RA populations.

This prospective observational study addresses these research gaps by systematically examining the impact of standard RA treatments on both clinical and psychological outcomes. By investigating the correlations between pharmacological interventions, disease activity, and psychiatric comorbidities such as anxiety and depression, the study aims to provide evidence supporting the integration of psychological evaluation and intervention into routine RA care. Ultimately, improving long-term clinical outcomes and QoL among RA patients.

Materials and Methods

This prospective observational study was conducted in the Department of Medicine at a tertiary care center in Central India over 1 year, with ethical clearance from the Institutional Scientific and Ethical Committee. The primary objective was to assess the impact of treatment on clinical and psychological outcomes in patients newly diagnosed with RA.

A sample size of 150 patients was calculated based on the regional prevalence of RA, estimated between 0.3% and 0.75% as reported in previous studies conducted in India,[4] along with consideration of statistical power and anticipated attrition. The study included newly diagnosed, untreated RA patients aged 18 to 60 years, classified according to the ACR/EULAR criteria, who were attending the rheumatology outpatient clinic and the Medicine departments and were willing to provide written informed consent. Both male and female patients were included.

Patients were excluded if they refused consent, had a prior diagnosis of RA with ongoing treatment, a pre-existing psychiatric illness, or a history of psychotropic medication use. Other exclusion criteria included pregnant or lactating women, patients with significant comorbid conditions that might affect mental health (such as severe cardiovascular, neurological, or endocrine disorders), and institutionalized individuals.

Eligible patients underwent detailed baseline assessments that included comprehensive clinical history, physical examination, and evaluation of disease severity using the Disease Activity Score 28 (DAS28). Psychological status was assessed using validated instruments: HDRS for depression and Hamilton Anxiety Rating Scale (HAMA) for anxiety. Baseline laboratory investigations included complete blood count (CBC), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), antinuclear antibody (ANA), anticyclic citrullinated peptide (Anti-CCP), liver function tests (LFT), and renal function tests (RFT). Radiological investigations, including chest X-ray, electrocardiography (ECG), and abdominal ultrasonography, were performed when clinically indicated.

Following baseline evaluation, patients received standard RA therapy, including DMARDs, with or without biologics and corticosteroids, individualized according to clinical guidelines.

Patients were reassessed after 6 months for clinical and psychological changes. Repeat assessments of DAS28, HDRS, and HAMA were performed during follow-up. All data from baseline and follow-up visits were documented systematically in a structured master chart for analysis.

All statistical analyses were performed using SPSS software, version 24.0. Microsoft Excel was used for data management and graphical representation. Continuous variables were summarized as mean ± standard deviation (SD) along with median and interquartile range (IQR), while categorical variables were expressed as frequencies and percentages. Data normality was assessed using the Shapiro–Wilk test. For within-group comparisons of DAS28, HDRS, and HAMA scores before and after DMARD therapy, both paired *t*-tests and Wilcoxon signed-rank tests were applied, depending on distribution. Categorical severity changes were analyzed using McNemar's test. Effect sizes were calculated as Cohen's *d* with 95% confidence intervals for continuous outcomes, and odds ratios (OR) with 95% confidence intervals, along with Cramer's V for categorical outcomes. A p-value of < 0.01 was considered statistically significant.

Of the initial cohort of 150 patients enrolled, 28 were lost to follow-up, and 6 additional patients were excluded for nonadherence to DMARD therapy, resulting in a final analyzed sample of 116 patients.

Results

A total of 116 newly diagnosed RA patients were included, with 85 (73.3%) females and 31 (26.7%) males, yielding a female-to-male ratio of 2.7:1. The study population showed a mean age of 48.5 ± 12.47 years. Almost half of the participants (48.2%) were between 31 and 50 years, followed by 41.3% in the 51–70 years age group, while only 8.6% were below 30 years and 1.7% above 70 years, confirming the predominance of RA in middle-aged women (Table 1).

At baseline, 65.3% of patients had depression, and 57.3% had anxiety. Patients demonstrated a mean HDRS of 15.9 ± 8.4 (median 15.0; IQR 10–22) and a mean HAMA of 11.7 ± 6.8 (median 11.0; IQR 7–16), consistent with moderate to severe psychological impairment. At baseline, the DAS28 was 5.28 ± 1.06 (median 5.20; IQR 4.6–6.0), indicating severe disease activity.

Table 1: Demographic characteristics of RA patients (N = 116). Age is presented both as a categorical distribution and as mean ± standard deviation (SD). Percentages are calculated based on the total sample size

Parameter	*Count (n)*	*Percentage (%)*
Gender		
Male	31	26.7%
Female	85	73.3%
Age groups (years)		
18–30	10	8.62%
31–50	56	48.2%
51–70	48	41.3%
>70	2	1.72%
Mean age (years ± SD)	48.5 ± 12.47	–

After 6 months of DMARD therapy, significant improvements were noted in both clinical and psychological parameters. The mean HDRS score decreased to 11.3 ± 6.4 [median 11.0 (IQR 6–16)], corresponding to a mean reduction of 4.62 points (95% CI 3.08–6.17, $p < 0.001$). Both the paired t-test ($t = 5.92$, $p < 0.001$) and the Wilcoxon signed-rank test ($W = 4487$, $p < 0.001$) confirmed this improvement, despite violations of the normality assumptions (Table 2). The analysis showed a moderate effect (Cohen's $d = 0.55$; rank biserial correlation = 0.58), reflecting a meaningful decline in depression severity. Categorical analysis supported these findings, with patients without depression nearly doubling from 16.5% to 31.2% and severe depression cases falling from 23.9% to 5.5%. Patients were almost eight times more likely to improve to "no/mild depression" following therapy (OR = 7.79, 95% CI 2.92–20.76; Cramer's V = 0.41, large effect).

Anxiety scores also showed improvement. The mean HAMA score reduced to 9.0 ± 5.4 [median 8.5 (IQR 5–12)], a mean reduction of 2.71 points (95% CI 1.36–4.05, $p < 0.001$). This change was statistically confirmed by both the paired t-test ($t = 3.98$, $p < 0.001$) and Wilcoxon signed-rank test (W = 3690, $p < 0.001$) (Table 2). The effect size was small-to-moderate (Cohen's $d = 0.37$; $r = 0.41$), indicating that while the reduction in anxiety was less pronounced compared to depression, it remained clinically relevant. Severe anxiety decreased from 7.3% to 1.8%, and the proportion of patients without anxiety increased from 27.5% to 40.4%. Patients were about three times more likely to improve to "no/mild anxiety" after treatment (OR = 2.83, 95% CI 1.12–7.15; Cramer's V = 0.21), representing a favorable trend even though McNemar's test did not reach conventional significance ($p = 0.067$) (Table 3).

Disease activity also showed significant improvement. The mean DAS28 declined to 4.60 ± 1.25 [median 4.58 (IQR 3.7–5.3)], with a mean reduction of 0.70 (95% CI 0.46–0.95, $p < 0.001$). This was confirmed by both the paired t-test ($t = 5.72$, $p < 0.001$) and Wilcoxon signed-rank test (W = 4621, $p < 0.001$) (Table 2). The effect size was moderate ($d = 0.53$; $r = 0.57$), indicating a clinically meaningful reduction in disease activity. Importantly, the greatest DAS28 improvements were observed among patients who also experienced marked psychological recovery: those transitioning from severe to no depression showed a 32.3% improvement in DAS28, while those with major reductions in anxiety improved by 30.8%. In contrast, patients with persistent severe psychological morbidity exhibited only minimal changes in DAS28, underscoring the negative impact of unresolved depression and anxiety on disease control.

Taken together, these results demonstrate that DMARD therapy not only reduced inflammatory disease activity but also provided significant psychological benefit, particularly in terms of depressive symptoms, with anxiety showing a favorable but less robust response.

Discussion

Rheumatoid arthritis (RA) is a chronic autoimmune disorder characterized by significant morbidity involving both physical disability and psychological distress, particularly depression and anxiety. This prospective observational study investigated the prevalence and impact of psychiatric comorbidities in newly diagnosed RA patients and examined the effect of disease-modifying antirheumatic drug (DMARD) therapy on both disease activity and psychological well-being.

In our study, the majority of participants were middle-aged (mean age: 48.5 ± 12.47 years) and predominantly female (73.3%). This demographic profile mirrors global epidemiological trends where RA predominantly affects women in their middle ages, as highlighted by Giblon et al. and Amaowei et al.[23,24] Similar female

Table 2: Effect of DMARD therapy on continuous clinical and psychological parameters in RA patients (N = 116). Values are presented as mean ± SD and median (IQR). Both parametric (paired Student's t-test, reported as t with df) and nonparametric (Wilcoxon signed-rank test, reported as W) were applied, as the Shapiro–Wilk test indicated violation of normality assumptions for all variables. Effect sizes are reported as Cohen's d with 95% CI for t-tests and rank biserial correlation (r) for Wilcoxon tests. All results indicate statistically significant and clinically meaningful improvement after 6 months of DMARD therapy

Parameter	*Baseline [mean ± SD; median (IQR)]*	*Post-DMARD [mean ± SD; median (IQR)]*	*Mean difference (95% CI)*	*t (df)*	*Wilcoxon (W)*	*p-value*	*Effect size (95% CI)*
DAS28	5.28 ± 1.06; 5.20 (4.6–6.0)	4.60 ± 1.25; 4.58 (3.7–5.3)	0.70 (0.46–0.95)	5.72 (115)	4621	<0.001*	Cohen's $d = 0.53$ (0.34–0.72); $r = 0.57$
HDRS (depression)	15.9 ± 8.41; 15.0 (10–22)	11.3 ± 6.43; 11.0 (6–16)	4.62 (3.08–6.17)	5.92 (115)	4487	<0.001*	Cohen's $d = 0.55$ (0.35–0.74); $r = 0.58$
HAMA (anxiety)	11.7 ± 6.80; 11.0 (7–16)	9.0 ± 5.40; 8.5 (5–12)	2.71 (1.36–4.05)	3.98 (115)	3690	<0.001*	Cohen's $d = 0.37$ (0.18–0.56); $r = 0.41$

*$p < 0.01$ considered statistically significant

Table 3: Effect of DMARD therapy on categorical severity levels of depression and anxiety ($N = 116$). Categorical severity levels of depression and anxiety were dichotomized as no/mild versus moderate/severe based on standard HDRS and HAMA cut-offs. McNemar's χ^2 test was used to assess paired categorical changes before and after DMARD therapy. Effect sizes are expressed as odds ratios (OR) with 95% confidence intervals, and Cramer's V. A significant and large effect was observed for depression, while anxiety showed a favorable but nonsignificant trend

Outcome	*Baseline n (%)*	*Post-DMARD n (%)*	*Change*	*McNemar's χ^2 (p)*	*Odds ratio (95% CI)*	*Cramer's V*
HDRS (depression)	No/mild: 50 (43.1%) Mod/severe: 66 (56.9%)	No/mild: 76 (65.5%) Mod/severe: 40 (34.5%)	+ 22.4% improvement	16.45 ($p < 0.001$*)	7.79 (2.92–20.76)	0.41
HAMA (anxiety)	No/mild: 80 (69.0%) Mod/severe: 36 (31.0%)	No/mild: 92 (79.3%) Mod/severe: 24 (20.7%)	+ 10.3% improvement	3.36 ($p = 0.067$)	2.83 (1.12–7.15)	0.21

*$p < 0.01$ considered statistically significant

predominance was also noted by Pezzato et al. (80% female), confirming consistency across populations regarding RA demographics.[25]

At baseline, we observed substantial psychiatric morbidity: 65.33% of RA patients had depression (HDRS ≥7), and 57.33% exhibited anxiety (HAMA ≥7). These findings align closely with the high depression prevalence (77%) and anxiety prevalence (69%) in Indian RA patients reported by Singh et al. and Amaowei et al., who found a high overall psychiatric comorbidity rate of approximately 71% among RA patients.[24,26] Moreover, Maldonado et al. reported a 42.9% depression prevalence among RA patients, associating it significantly with higher disease activity and disability,[27] reinforcing the critical link between psychiatric morbidity and RA disease severity.

Our baseline clinical assessments demonstrated moderate-to-high psychological distress (HDRS: 15.9 ± 8.4, HAMA: 11.7 ± 6.8) alongside high disease activity (DAS28: 5.28 ± 1.06). These results corroborate findings from Moudi et al., who also reported significant correlations between DAS28 scores and psychiatric morbidity, emphasizing the intertwined nature of mental health and physical symptoms in RA.[28] Englbrecht et al. similarly stressed that disease activity measures alone are insufficient to capture depressive symptoms, advocating regular depression screening alongside RA assessments.[29]

After 6 months of DMARD therapy, significant clinical and psychological improvements were evident. Mean DAS28 scores decreased significantly (5.3 ± 1.04 to 4.6 ± 1.3; $p < 0.001$), alongside marked improvements in depression (HDRS: 28.93% improvement) and anxiety (HAMA: 23.08% improvement). These findings align with Panjrattan et al., who similarly highlighted that effective RA management significantly reduces psychiatric symptoms by decreasing pain and disease activity.[30] Our observations support the proposition by Ionescu et al. that effectively managing RA disease activity can concurrently reduce depression severity, thus potentially enhancing overall prognosis.[22]

In our cohort, the shift toward milder psychiatric symptoms after treatment was particularly notable. Severe depression prevalence markedly decreased (23.85% to 5.50%), and the proportion of patients free from depression nearly doubled. Severe anxiety similarly declined (7.34% to 1.83%), while anxiety-free individuals increased substantially (27.52% to 40.37%). This underscores that effective RA treatment can yield substantial psychological benefits, reinforcing the dual role of inflammation in physical and mental health symptoms in RA, as previously discussed by Margaretten et al., who described inflammation as an underlying biological mechanism linking RA with depressive symptoms.[31]

Detailed subgroup analyses further illustrated a robust correlation between psychiatric improvement and reduced RA disease activity. The greatest improvements in DAS28 scores were seen among patients transitioning from severe psychiatric symptoms to no symptoms (32.32% for depression, 30.77% for anxiety). Conversely, minimal clinical improvement was observed among patients with persistent severe psychological symptoms, emphasizing how unresolved depression and anxiety can negatively impact RA management. Similar observations were reported by Fakra et al., who noted that untreated depression adversely affects disease activity, treatment outcomes, and overall quality of life in RA patients.[11]

The hypothesis that systemic inflammation contributes directly to psychiatric morbidity, particularly depression, provides a plausible biological explanation for these clinical observations. Maldonado et al. emphasized the association between high disease activity, increased joint pain, and higher depression rates.[27] Further, Englbrecht et al. highlighted that inflammatory pathways likely play a substantial role in depressive symptomatology among RA patients.[29] Similarly, Soskin et al. supported the inflammatory hypothesis of depression, indicating proinflammatory cytokines and immune dysregulation as significant contributors to depression pathogenesis, particularly relevant to chronic inflammatory diseases such as RA.[32] Our study's observation of concurrent improvements in disease activity and psychological symptoms following DMARD therapy—known for its anti-inflammatory effects—further substantiates this theory, proposing inflammation as a critical common factor in both RA and depression.

Despite notable improvements, residual depression and anxiety symptoms persisted in some patients, highlighting the necessity for adjunctive psychiatric interventions. Pezzato et al. similarly reported underdiagnosis and inadequate management of depression in RA patients, despite clear associations with poorer clinical outcomes, emphasizing that addressing psychological comorbidities is crucial for comprehensive disease management.[25] Fakra further reinforced the importance of integrated psychiatric care within RA management to enhance clinical outcomes.[11]

Our findings advocate for regular psychiatric screening, combined with comprehensive RA treatment strategies, potentially including pharmacological, psychotherapeutic, and supportive interventions, to enhance clinical outcomes. Margaretten et al. underscored the multifaceted nature of depression in RA, suggesting that an integrated approach addressing socioeconomic, biological, and psychological factors is necessary for effective management.[31]

Conclusion

This study demonstrates that effective RA treatment with DMARDs significantly

improves both clinical and psychological outcomes in RA patients, reinforcing the interconnected nature of inflammation, mental health, and disease activity. Routine psychiatric screening and integrated mental health interventions alongside traditional RA therapy are recommended to optimize treatment outcomes and enhance patient quality of life.

Limitations

This study had certain limitations, including a relatively short follow-up period and a moderate sample size, which may have affected the generalizability of the findings. Additionally, psychological assessments relied on self-report scales, which may introduce subjective biases. Larger, multicentric studies with longer follow-up periods and objective psychiatric evaluations are recommended for future research.

Acknowledgments

The authors sincerely thank all participants who volunteered for this study, as well as the medical and nursing staff who facilitated data collection and patient care throughout the research period. The authors gratefully acknowledge the contribution of Dr Tasneem Hussain (MD, Pharmacology, MGM Medical College, Indore) for her assistance with statistical analysis, critical interpretation of data, and support with medical writing.

Authors Contribution

All authors contributed to the concept and design, acquisition, analysis, and interpretation of data, drafting of the manuscript, and critical review of the manuscript for important intellectual content.

Conflict of Interest

None.

Data Availability

The data underlying the study's findings can be obtained from the corresponding author upon reasonable request.

Source of Support

None.

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