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CLINICAL IMPACT OF DEPRESSION ON TREATMENT OUTCOMES IN RHEUMATOID ARTHRITIS: A LITERATURE REVIEW

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ABSTRACT

Depression is a common comorbidity in rheumatoid arthritis, however we still don't know enough about how it affects treatment outcomes. This systematic review aims to assess the clinical influence of depression on disease activity, remission rates, pharmacological treatment response, patient-reported outcomes, and treatment adherence behaviour in individuals with rheumatoid arthritis. A thorough literature review was performed using PubMed, Scopus and Web of Science. We focused on studies published from 2020 to 2026 that evaluated depressive symptoms through validated instruments and reported at least one treatment-related outcome. The results consistently indicated that comorbid depression correlated with greatly elevated composite disease activity scores. It was mostly influenced by subjective rather than objective components. Patients exhibiting depressive symptoms were less likely to attain remission and demonstrated diminished responses to both conventional synthetic and biologic disease-modifying antirheumatic drugs. Depression was also found to be a strong independent predictor of worse health-related quality of life, more pain, more functional disability and less adherence to treatment. These findings underscore the necessity for regular depression screening in rheumatology practice and advocate for the incorporation of mental health interventions into the management of rheumatoid arthritis to enhance treatment outcomes.

KEYWORDS

Arthritis, Rheumatoid, Depression, Treatment Outcome, Medication Adherence, Patient Reported Outcome Measures

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Introduction

Rheumatoid arthritis (RA) is a long-term, systemic autoimmune disease that causes ongoing inflammation of the synovial membrane, damage to the joints over time and major loss of function. About 0.5–1% of people around the world have it and it is more common in women and older people [1]. RA is becoming more and more known as a multisystem disorder with a lot of extra-articular and psychological effects, in addition to its joint symptoms. Among the psychiatric comorbidities associated with rheumatoid arthritis (RA), depression is the most commonly observed condition, with prevalence estimates varying from 13% to over 40%, contingent upon the studied population and the diagnostic tools utilized [2].

Depression and RA do not just happen to be present at the same time. Some researchers think that this two-way relationship is caused by shared pathophysiological mechanisms, such as dysregulation of pro-inflammatory cytokines like interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis and changes in neurotransmitter metabolism [1]. Chronic pain, limited mobility, fatigue and the mental and social effects of living with a disabling condition all make RA patients more likely to develop and keep having depressive symptoms [2]. On the other hand, depression can make pain feel worse, make it harder to cope and make it harder to be active, all of which can make RA worse [3].

The treat-to-target (T2T) principle guides modern RA management. This principle says that drugs should be given early and aggressively in order to achieve clinical remission or, at the very least, low disease activity. This method uses composite disease activity measures like the Disease Activity Score in 28 joints (DAS28) and requires regular monitoring and changes to treatment [4]. These composite indexes, on the other hand, include patient-reported components like tender joint counts and global health assessments, which may be more affected by depressive symptoms than by actual inflammatory activity [5]. This makes it possible that depression could mess up disease activity tests, which could lead to the wrong increase in immunosuppressive therapy, or to the wrong impression of treatment failure when there is no real inflammatory disease [3].

Zhang et al. did a systematic review and meta-analysis that showed a strong link between depression and higher disease activity and a lower quality of life related to health in RA patients. This shows how important this comorbidity is in the clinic [4]. Narrative and systematic reviews have also shown that depressive symptoms can make it harder to stick to treatment plans, make disease-modifying antirheumatic drugs (DMARDs) and biologic agents less effective, and make people use more healthcare [3]. Studies that look at entire populations have also shown that the number of RA patients with depression and anxiety has gone up over the past few decades, which suggests that this is becoming a bigger clinical problem [6].

Even though more and more evidence is coming in, there is still a big gap in the literature when it comes to a full synthesis of data that looks at how depression directly affects treatment outcomes for all types of RA therapies, such as conventional synthetic DMARDs (csDMARDs), biologic DMARDs (bDMARDs), and targeted synthetic DMARDs (tsDMARDs). Most of the reviews that are out there have looked at either how common depression is in people with RA or how RA treatment affects depressive symptoms. They have not looked at the opposite relationship. Also, there are a lot of various kinds of depression assessment instruments and study designs in the literature, which implies that a comprehensive, systematic examination is necessary.

Aim

The objective of this systematic review was to assess the clinical influence of depression on treatment outcomes in patients with rheumatoid arthritis, including disease activity, remission rates, pharmacological treatment response, patient-reported outcomes, and treatment adherence.

Methodology

We did a thorough search of articles published in the last ten years, with a focus on studies from 2020 to 2026, using several electronic databases such as PubMed/MEDLINE, Scopus and Web of Science. This was the basis for this systematic review. The keywords used in the search were rheumatoid arthritis, depression, depressive symptoms, treatment outcomes, disease activity, remission, treatment response, adherence, and patient-reported outcomes. We also looked at the reference lists of the articles we found and relevant reviews to find more studies that met the criteria. We only included peer-reviewed, English-language articles that involved adult patients with rheumatoid arthritis, used validated tools to measure depressive symptoms and reported at least one treatment-related outcome. We used the Joanna Briggs Institute Critical Appraisal tools and the Newcastle-Ottawa Scale to rate the quality of the studies. The analysis uses the most recent clinical findings, observational data, post hoc analyses of randomised controlled trials, and meta-analytic evidence to give a thorough picture of what we currently know about how depression affects treatment outcomes in rheumatoid arthritis and to point the way for future research and practice in this area.

Results

Prevalence and Measurement of Depression Across Included Studies

The studies that were included showed a wide range of rates of depressive symptoms in RA patients. This was due to differences in the populations studied, the locations of the studies, and the diagnostic tools used. Ionescu et al. did a cross-sectional study in Romania and found that about 34% of RA patients had clinically significant depressive symptoms when they were given the Patient Health Questionnaire-9 (PHQ-9). The rates were higher in people who had the disease for a longer time and had more active disease [7]. The same research group did another study using both the PHQ-9 and the Hospital Anxiety and Depression Scale (HADS) and found that more than 30% of people had depression. They also found a strong link between depressive symptoms and disease activity as measured by DAS28 [8].

Pham et al. found that almost 40% of RA patients in a Vietnamese group met the DSM-5 criteria for depressive disorders. Most of these patients were classified as having mild to moderate depression [9]. The authors pointed out that being female, unemployed, and experiencing more pain were all linked to having depressive disorders in their population. Giblon et al. did a population-based study in the United States that looked at changes over time in anxiety and depression among people with RA. They found that the rates of both conditions have gone up a lot over the past few decades, with depression rates reaching about 20% in the most recent group [6].

Fragoulis et al. looked at data from an early RA inception cohort in Scotland and found that about 25% of patients were depressed at the time of diagnosis, according to the HADS [10]. Notably, having depressive symptoms at the beginning was linked to having a lower socioeconomic status, more pain, and worse functional capacity, but not to markers of inflammation in the blood. This observation suggests that psychosocial and

symptom-related factors may play a bigger role in the burden of depression in early RA than the underlying inflammatory disease activity [10].

The fact that there are so many different ways to measure depression is a major methodological problem. The PHQ-9, the HADS, and the Beck Depression Inventory (BDI) were the most common tools used in the studies that were included. Structured clinical interviews based on DSM criteria were used in fewer studies. Matcham et al. have already pointed out that different tools give very different estimates of how common something is because they have different cut-off points, include somatic items that are similar to RA symptoms, and are not the same as screening tools and diagnostic assessments [5]. This difference makes it hard to compare studies directly and shows how important it is to have standardised ways to measure depression in RA research.

Different studies also looked at depression at different times in relation to when RA treatment started. Some studies looked at depressive symptoms when RA was first diagnosed or when a new treatment plan began, while others looked at depression at different points in time during the course of the disease. This temporal heterogeneity is important because the number of people with depression may change over time as RA progresses, as they respond to treatment, and as they adapt to their new situation [11].

Impact of Depression on Objective Disease Activity and Remission

The correlation between depression and objective indicators of disease activity in rheumatoid arthritis (RA) has been the subject of multiple studies, consistently indicating that depressive symptoms correlate with elevated composite disease activity scores. In their systematic review and meta-analysis, Zhang et al. showed that RA patients with comorbid depression had significantly higher DAS28 scores than those without depression. This relationship held true across various study designs and geographic regions [4]. The meta-analytic results revealed a moderate yet clinically significant effect size, supporting the hypothesis that depression independently exacerbates disease activity indexes.

Ionescu et al. found that depressed RA patients had much higher DAS28-CRP scores than RA patients who were not depressed, even after controlling for demographic and clinical factors [7]. The disparity in disease activity scores was predominantly influenced by the subjective elements of DAS28, such as tender joint count and patient global assessment, rather than by objective inflammatory indicators like C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR) [7]. This finding supports the hypothesis that depression may exaggerate composite disease activity measures by enhancing the perception and reporting of symptoms rather than through a true intensification of the inflammatory process.

Henkemans et al. investigated the influence of depression on remission rates through a multicenter study involving patients with rheumatoid arthritis and psoriatic arthritis, treated according to a standardised treat-to-target strategy [12]. Their study showed that patients who were depressed or anxious at the start of the study were much less likely to reach DAS28 remission after 12 months. The failure to achieve remission in depressed patients continued even after controlling for baseline disease activity, treatment regimen, and demographic factors, indicating that depression serves as an independent obstacle to reaching therapeutic objectives in RA [12].

Matcham et al. also found that RA patients who had more depressive symptoms at the start of the study had higher disease activity scores over time. This shows that depression has a long-term effect on disease activity, not just a short-term one [5]. Dagli et al.'s narrative review backed up these findings and came to the same conclusion: depression is always a sign of worse disease outcomes, and its effects go beyond what patients say to affect the overall course of RA management [3].

Fragoulis et al. also showed that having depressive symptoms early on in RA was linked to higher disease activity at the time of presentation. However, this link was weaker when only objective inflammatory markers were examined [10]. This pattern held true across studies, which suggests that depression affects disease activity scores in part by making symptoms worse. Some data, however, suggest that depression may also have direct biological effects on inflammation through pro-inflammatory cytokine pathways. Nevertheless, this effect seems to be smaller than the subjective component [1].

Influence of Depression on Pharmacological Treatment Response

Researchers have looked into how comorbid depression affects the drug treatment of RA in a number of different ways. Dagli et al. looked at the evidence that was available on how depression affects treatment response in inflammatory arthritis and found that depressive symptoms were always linked to a lower chance of getting a clinically meaningful response to DMARDs [3]. Their narrative synthesis showed that depressed patients were more likely to be labelled as non-responders according to the European Alliance of Associations for Rheumatology (EULAR) response criteria, no matter what DMARD regimen was used.

Citera et al. did a post hoc analysis of data from several phase 3 and phase 3b/4 randomised controlled trials of tofacitinib, a Janus kinase (JAK) inhibitor, in RA patients who were either likely to be depressed or anxious or not [13]. Their results showed that tofacitinib improved clinical outcomes in both groups, but patients with likely depression or anxiety had a smaller improvement in ACR response rates and DAS28 scores than those without psychiatric comorbidity [13]. However, the absolute treatment benefit of tofacitinib was still statistically significant in the depressed subgroup. This shows that JAK inhibitors are still effective even when someone is psychologically distressed.

Several studies have indicated that depression diminishes the response to biologic DMARDs. Henkemans et al. discovered that rheumatoid arthritis patients undergoing bDMARD treatment within a treat-to-target framework, who also had comorbid depression, were markedly less likely to attain sustained remission and more often needed treatment escalation [12]. Tanski et al. assessed RA patients undergoing biologic therapy at a single institution and found that individuals exhibiting elevated depressive symptoms demonstrated lesser enhancements in disease activity and functional outcomes, despite similar baseline inflammatory markers [14].

The mechanisms responsible for the diminished treatment response in depressed RA patients are probably multifactorial. Impaired treatment adherence, constitutes a major contributing factor. Moreover, depression-related modifications in the immune-inflammatory environment, characterized by consistently heightened levels of pro-inflammatory cytokines like IL-6, may partially mitigate the immunosuppressive effects of DMARDs and biologic agents [1]. Behavioral factors, such as decreased physical activity, disturbed sleep, and ineffective pain management, may worsen treatment outcomes in this population [11].

The treat-to-target strategy, which depends on regular evaluations and standardized treatment modifications, may be especially susceptible to the confounding influences of depression. When composite disease activity scores stay high because of the subjective effects of depressive symptoms, doctors may give more treatment than is needed or think that the treatment isn't working when it really is [3]. This phenomenon has significant ramifications for patient safety and the allocation of healthcare resources.

Depression and Patient-Reported Outcomes (PROs)

Patient-reported outcomes (PROs) are becoming more and more important for establishing how well RA treatments work. They are also areas where depression has significant impact. Several studies have looked into the link between depression and health-related quality of life (HRQoL) in RA patients, and they all concluded that it was a negative one. Rojas-Gualdrón et al. used the EQ-5D instrument to assess HRQoL in a cohort of seropositive RA patients, revealing that self-reported depression and anxiety were the most significant predictors of HRQoL decline, exceeding the effects of pain, mobility restrictions, and challenges in daily activities [15]. This finding highlights the unequal effect of psychological distress on the subjective well-being of RA patients.

Alwhaibi reviewed data from a national health survey and found that RA patients with comorbid depression displayed considerably lower HRQoL scores in both physical and mental health domains compared to those without depression [16]. The extent of HRQoL decline linked to depression was similar to or surpassed that resulting from increased disease activity, indicating that the psychological impact of depression may parallel the inflammatory impact of RA on overall patient well-being [16].

Pain, a principal symptom of RA and a major concern for patients, is significantly affected by comorbid depression. Abdelrahman showed that depressive symptoms were strongly and independently linked to higher pain intensity scores in RA patients, even when taking into account objective measures of disease activity and joint damage [17]. The enhancement of pain perception by depression is widely acknowledged in the pain literature and is believed to involve central sensitization, modified descending pain modulation, and cognitive patterns of catastrophizing [1]. In the context of RA, this pain amplification may result in a gap between objectively assessed inflammatory control and the patient's subjective perception of their disease.

Functional disability, as assessed by the Health Assessment Questionnaire (HAQ), is another patient-reported outcome (PRO) domain negatively influenced by depression. Rizaj and Kelmendi assessed RA patients undergoing treatment with biologic agents and discovered that individuals exhibiting elevated depressive symptom scores reported substantially greater functional limitations on the HAQ, despite attaining similar levels of inflammatory disease control [18]. Tanski et al. confirmed this finding, showing that depression was a significant, independent predictor of HAQ scores in RA patients undergoing biologic treatment [14].

Fatigue, a common and severe symptom experienced by RA patients, is further intensified by comorbid depression. Abdelrahman discovered that sleep quality, strongly associated with depression and fatigue, was substantially compromised in RA patients exhibiting depressive symptoms [17]. The interaction between depression, sleep disturbance, and fatigue establishes a self-perpetuating cycle that may prove challenging to interrupt with anti-rheumatic therapy alone, underscoring the necessity for integrated management strategies.

A significant and clinically relevant finding across the included studies is the divergence between objective improvements in inflammatory parameters and subjective patient evaluations. Numerous studies have shown that depressed RA patients may not notice or report clinical improvement, even when laboratory markers of inflammation return to normal and joint swelling decreases objectively [3]. This discrepancy has major significance for the treat-to-target paradigm, as the sustained elevation of patient-reported elements within composite disease activity scores may lead to unwarranted treatment escalation in patients whose inflammatory disease is sufficiently managed [4].

Treatment Adherence, Persistence, and Healthcare Utilization

Adherence to treatment and persistence are very important for long-term outcomes in RA. Depression has been shown to be a major barrier to taking medication as prescribed. Khadour et al. did a qualitative survey to find out what factors affect depression and anxiety in RA patients. They found that low motivation, hopelessness regarding how well treatment would work, cognitive problems that make it hard to manage medication, and fear of side effects amplified by negative cognitive biases were all barriers to treatment adherence [11].

Fakra and Marotte looked at the two-way link between RA and depression and pointed out that RA patients who are depressed are much more likely not to follow their prescribed DMARD regimens [1]. The authors said that not following through with treatment is often caused by more than one thing. For example, people may stop treatment on purpose because they think it isn't working, or they may forget things or get disorganized because of cognitive impairment caused by depression [1].

Treatment persistence, which is the length of time someone stays in therapy without stopping, is also adversely affected by depression. Dagli et al. found that depressed people with inflammatory arthritis were more likely to stop treatment early than non-depressed patients. This was true for both csDMARDs and bDMARDs [3]. Stopping treatment too soon not only lowers the chances of long-term disease control, but it also raises the risk of disease flare-ups and permanent joint damage.

There are big differences in how RA patients with and without comorbid depression are treated. Ionescu et al. found that depressed RA patients went to the doctor more often, had higher rates of the emergency department attendance, and were hospitalized more frequently than non-depressed patients, even after taking into account the severity of their illness [2]. Alwhaibi also found that RA patients with depression used more healthcare resources, such as more analgesic medications, more visits to specialists, and higher overall healthcare costs [16].

The economic consequences of increased healthcare utilization among depressed RA patients are major. The direct costs of extra doctor visits, medications, and hospital stays, as well as the indirect costs of lower workplace productivity and higher disability, put a lot of stress on both patients and healthcare systems [15]. These results make a strong case for finding and treating depression early in patients with RA from both health and economic point of view.

Zhang et al. also pointed out that psychological treatments aimed at relieving depressive symptoms in RA patients may have long-term benefits for treatment adherence and healthcare use, even though there isn't much evidence for specific types of interventions [19]. Adding mental health support to rheumatology care pathways could be a cost-effective way to deal with the many ways that depression may affect RA treatment outcomes.

Discussion

The results of this systematic review consistently show that depression has a clinically significant negative effect on many areas of RA treatment outcomes. Patients with RA who are depressed have higher composite disease activity scores, are less likely to go into remission, respond more poorly to medications, report worse pain, fatigue, functional disability, and quality of life. They are also less likely to stick to their treatment and they use more healthcare resources. These links were seen in a variety of study designs, locations, and treatment types, which makes the overall conclusions extremely convincing.

It's not clear how depression affects the results of RA treatment, but it's likely that it does so through both biological and psychobehavioral pathways. From a biological point of view, depression is linked to the constant activation of the body's immune system, higher levels of pro-inflammatory cytokines like IL-6, TNF-α, and interleukin-1 beta (IL-1β) and problems with the HPA axis [1]. These changes in the immune system may keep or worsen the inflammatory environment in RA, which may make immunosuppressive treatments less effective [2]. Changes in neurotransmission related to depression, such as changes in serotonergic and noradrenergic neurotransmission, may also cause central sensitization and enhanced pain processing, which could make subjective measures of disease activity seem higher [3].

From a psychobehavioral point of view, depression is linked to lower self-efficacy, less physical activity, worse sleep quality, ineffective ways of dealing with pain, and more catastrophizing [17]. All of these things make it harder for the patient to actively manage their disease, follow complicated treatment plans, and do activities that help them get better. The resulting lack of behavioural engagement could make even the best prescribed drug treatments less effective [11].

One very important clinical implication of these findings has to do with how to understand composite disease activity measures in depressed RA patients. The DAS28 and similar indexes include parts that patients report, which can be affected by depressive symptoms. Several of the studies that were included showed that the link between depression and high DAS28 scores was mostly caused by subjective factors, while objective inflammatory markers were not as affected [7]. This observation is directly related to the treat-to-target strategy because doctors may wrongly think that persistently high disease activity scores are due to poor inflammatory control when the real cause is psychological distress that hasn't been addressed [12]. If this happens, the patient may have to go through more treatment than necessary, be exposed to the risks of stronger immunosuppressive drugs, and not get to the bottom of what is really causing their symptoms.

The results of this review also show how important it is for doctors to regularly check for depression in RA patients. Even though more and more people are becoming aware of how common and serious depression is in individuals with RA, systematic screening is still not done consistently [6]. The PHQ-9 and HADS are both validated, short, and easy-to-use tools for screening for depression in rheumatology settings. They could easily be added to regular clinical assessments [5]. If depressive symptoms were found early, the person could be sent for psychological or psychiatric help right away, which could improve both their mental health and their response to RA treatment.

There are a few problems with this review that need to be pointed out. First, the studies that were included used different definitions and measurement tools for depression, which made it hard to compare them directly and made it impossible to do a formal meta-analysis in some outcome domains. Second, most of the studies that were included were observational, which makes it hard to say what caused the relationship between depression and treatment outcomes. Third, we can't rule out the possibility that unmeasured variables, such as socioeconomic status, comorbid medical conditions, and a history of mental illness, could still be causing problems. Fourth, publication bias may have affected the evidence base that is out there. Studies that find a strong link between depression and worse outcomes may be more likely to be published than studies that find no link.

Future research should focus on prospective long-term studies that use standardized methods to measure depression in order to understand the relationship between depression and RA treatment outcomes over time. We urgently need randomized controlled trials to see how treating depression, whether with drugs, therapy, or a combination of the two, affects RA disease activity and treatment response. It would be helpful for healthcare policy and resource allocation to have studies that look at how cost-effective integrated care models are that include mental health support within rheumatology care pathways

Conclusions

This systematic review offers compelling evidence that depression is a crucial and potentially alterable factor that negatively influences treatment outcomes in rheumatoid arthritis. Depression negatively impacts disease activity assessment, remission attainment, pharmacological treatment response, outcomes reported by patients, and treatment adherence. Depressive symptoms may distort composite disease activity measures, resulting in clinical decisions that fail to accurately represent the underlying inflammatory condition.

Routine screening for depression should be incorporated into standard rheumatological care for all RA patients, involving validated instruments such as the PHQ-9 or HADS. Clinicians must recognize that consistently high disease activity scores alongside comorbid depression may indicate psychological distress rather than insufficient inflammatory control, and treatment decisions should be guided by this understanding.

The incorporation of psychiatric and psychological care into rheumatology practice signifies a vital advancement in the holistic management of rheumatoid arthritis. Treating depression along with inflammatory disease could make not only mental health outcomes better, but also improve disease activity control, treatment response, functional capacity, and overall quality of life. Subsequent research needs to concentrate on creating and assessing integrated care models that systematically and empirically address the complex connection between depression and rheumatoid arthritis.

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