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SLEEP DISTURBANCES AND FATIGUE IN ANKYLOSING SPONDYLITIS: A NARRATIVE REVIEW OF PREVALENCE, MECHANISMS, CLINICAL IMPACT AND MANAGEMENT

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ABSTRACT

Sleep disturbances and fatigue are common and burdensome manifestations of ankylosing spondylitis (AS). They are strongly associated with overall disease burden and impaired health-related quality of life. This narrative review summarises current evidence on their prevalence, underlying mechanisms, clinical consequences, assessment and management. Sleep disturbances affect approximately half of patients with AS, while clinically significant fatigue is reported by a similar proportion of patients across axSpA populations. Both symptoms are associated with greater pain, higher disease activity, impaired physical function, psychological distress, reduced social participation, and poorer quality of life. Fatigue may also affect selected dimensions of occupational functioning. Their pathogenesis is multifactorial. Inflammatory signalling may contribute to both symptoms, with TNF- α having the clearest established role in sleep regulation and broader inflammatory pathways, including TNF- α and IL-17, potentially contributing to fatigue. Pain, restricted mobility, functional impairment, anxiety, depression, chronic insomnia, and coexisting sleep disorders may also play important roles. Anti-inflammatory treatment can improve fatigue and patient-reported sleep outcomes, although objective sleep findings remain inconsistent and residual symptoms may persist despite improvements in disease activity. Exercise, physiotherapy, patient education, and appropriate management of insomnia and coexisting sleep disorders may complement pharmacological treatment. Sleep quality and fatigue should therefore be assessed directly as distinct patient-reported domains. Further longitudinal and interventional studies using standardised outcome measures are needed to clarify causal relationships, determine symptom-specific treatment effects, and support more individualised management.

KEYWORDS

Ankylosing Spondylitis, Sleep Disturbances, Fatigue, Quality of Life, Patient-reported Outcomes, Clinical Management

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Introduction

Ankylosing spondylitis (AS), corresponding to the radiographic form of axial spondyloarthritis (r-axSpA), is a chronic inflammatory rheumatic disease primarily affecting the axial skeleton [1,2]. It is characterised by chronic inflammation, pathological new bone formation, and progressive structural damage, leading to chronic back pain, spinal stiffness, reduced mobility, and functional impairment [1]. AS typically begins before the age of 45 years, with symptom onset most commonly occurring in early adulthood [3]. Its estimated prevalence ranges from approximately 0.07% to 0.32% across continents and varies according to geographic region and population HLA-B27 prevalence [4]. Although AS has historically been considered a predominantly male disease, sex-related differences in clinical presentation and historical diagnostic bias may contribute to delayed diagnosis and under-recognition in women [5].

Over the past two decades, the introduction of biologic and targeted synthetic disease-modifying antirheumatic drugs has expanded therapeutic options and improved the control of symptoms and disease activity in patients with AS [6]. Current ASAS–EULAR recommendations emphasise individualised treatment, shared decision-making, and regular monitoring of disease activity, function, comorbidities, and treatment response [6]. Nevertheless, substantial symptom burden may persist despite improvements in disease control, highlighting the importance of incorporating patient-reported outcomes into the comprehensive assessment of AS [6,7].

Sleep disturbances and fatigue are among the most frequent and burdensome patient-reported manifestations of AS and are strongly associated with impaired daily functioning and health-related quality of life [8–11]. Despite growing recognition of their clinical importance, differences in definitions, assessment methods, and study populations continue to make comparisons across studies difficult [8,9]. This narrative review summarises current evidence regarding the prevalence, underlying mechanisms, clinical consequences, assessment, and management of sleep disturbances and fatigue in patients with AS.

Methods

A targeted narrative literature search was carried out in PubMed and Google Scholar. The search included English-language publications on sleep disturbances and fatigue in ankylosing spondylitis and, where relevant, in broader axial spondyloarthritis populations. Studies addressing prevalence, underlying mechanisms, clinical impact, assessment, health-related quality of life, and management were considered.

The review included original research, systematic reviews, meta-analyses, psychometric studies, and clinical practice guidelines. Greater weight was given to recent publications and higher-level evidence. Older landmark studies were also retained when they provided important epidemiological, clinical, pathophysiological, or measurement data.

The selected publications were reviewed and synthesised narratively. Particular attention was given to the relationships between sleep disturbances, fatigue, disease activity, physical functioning, psychological well-being, and health-related quality of life. No formal risk-of-bias assessment or quantitative synthesis was performed.

Results

1. Pathophysiology

The pathophysiology of ankylosing spondylitis (AS) involves a complex interplay between genetic predisposition and immune-mediated inflammation [2,12]. Inflammatory changes primarily affect the sacroiliac joints, spine, and entheses. Enthesitis is regarded as a hallmark feature of the disease and may initiate local tissue damage and repair responses at sites exposed to mechanical stress [2].

Genetic susceptibility plays a central role in disease development. HLA-B27 shows the strongest known association with AS [12,13]. Several immune pathways participate in axial inflammation, particularly those mediated by tumour necrosis factor- α (TNF- α) and interleukin-17, whereas the role of interleukin-23 in established axial disease remains less clearly defined [2,12]. HLA-B27 may contribute to pathogenesis through several non-mutually exclusive mechanisms, including altered peptide presentation, aberrant protein folding, and modulation of innate and adaptive immune responses [13].

Structural progression results from the interaction between inflammatory injury, tissue repair, and osteogenic responses [2,14]. At vertebral and enthesal sites, these processes may promote pathological new bone formation, leading to syndesmophyte development, spinal ankylosis, and progressive restriction of mobility [12,14]. Although spinal inflammation is associated with subsequent structural damage, radiographic progression is not fully explained by contemporaneous inflammatory activity and may continue after active inflammatory lesions have subsided [14].

The immune mechanisms involved in axial inflammation are also associated with extra-musculoskeletal manifestations, particularly acute anterior uveitis, psoriasis, and inflammatory bowel disease [2,12]. Together, genetic susceptibility, immune activation, tissue repair, and pathological bone remodelling shape the heterogeneous clinical and structural phenotype of AS.

2. Disease Burden

Although inflammation and structural progression are the principal pathological processes in AS, they do not fully explain the burden experienced by patients in everyday life [7]. This discrepancy has led to increasing recognition of the importance of patient-reported outcomes (PROs), which complement clinical, laboratory, and imaging assessments by addressing aspects of health that are not fully reflected in objective measures [6,7]. PROs encompass multiple domains, including pain, fatigue, sleep quality, physical functioning, and health-related quality of life, providing a comprehensive evaluation of overall disease burden [7].

Despite major advances in treatment and improved control of disease activity with biologic and targeted synthetic disease-modifying antirheumatic drugs, a clinically relevant burden of symptoms may persist [6]. Studies of patients who meet remission or low-disease-activity criteria have shown that many continue to report residual symptoms and impaired health status [15,16]. These findings indicate that achieving conventional disease activity targets does not necessarily correspond to complete symptom resolution or full restoration of physical function and quality of life [15,16].

Fatigue and sleep disturbances are important components of the overall disease burden [8–11]. Increasing evidence indicates that both symptoms are common and clinically relevant manifestations of AS, frequently coexist, and may influence one another through complex biological and psychosocial mechanisms [8–11].

3. Sleep Disturbances

3.1. Prevalence and Characteristics

Sleep disturbances are a common and clinically important feature of ankylosing spondylitis. They affect an estimated 53% of patients, although this estimate varies depending on the study population and assessment methods [8,17]. Sleep impairment encompasses a broad spectrum of disturbances, including difficulties initiating and maintaining sleep, fragmented and non-restorative sleep, as well as reduced sleep efficiency [17,18]. Consequently, patients with AS consistently report poorer overall sleep quality than healthy individuals, reflected by significantly higher global Pittsburgh Sleep Quality Index (PSQI) scores [18].

Beyond impaired sleep quality, specific sleep disorders have also been reported in patients with AS. Evidence regarding an increased prevalence of obstructive sleep apnoea remains inconsistent [19,20], whereas restless legs syndrome appears to be more common in patients with AS than in healthy controls [21,22].

Polysomnographic studies have reported altered sleep parameters in patients with AS, including reduced sleep efficiency, increased nocturnal awakenings, and changes in sleep architecture [18,23].

3.2. Mechanisms and Contributing Factors

Sleep disturbances in ankylosing spondylitis (AS) arise from a complex interplay between inflammatory activity, disease-related manifestations, functional impairment, and psychological comorbidities rather than nocturnal pain alone [10,11,17,18].

Among the inflammatory mediators involved in AS, TNF- α has the clearest established role in sleep regulation [24–26]. Persistent TNF- α signalling may disrupt normal sleep architecture during chronic inflammatory states [24–26]. Anti-TNF therapy has been associated with improved subjective sleep quality, whereas objective polysomnographic findings have been mixed [23,27].

Beyond inflammatory pathways, disease-related symptoms constitute another major contributor to disturbed sleep. Pain is consistently identified as one of the strongest determinants of poor sleep quality in AS [10,17,18]. Unlike mechanical pain, inflammatory spinal pain typically worsens during the second half of the night, frequently causing repeated awakenings and preventing uninterrupted sleep [2,6,17]. Prolonged morning stiffness may further reflect nocturnal disease activity, while restricted spinal mobility, functional impairment, and discomfort during changes in body position may interfere with comfortable positioning and sleep continuity [10,17,28].

Psychological factors represent another important component of this multidimensional model. Anxiety and depressive symptoms occur more frequently in patients with AS than in the general population [29]. They are also associated with poorer sleep quality, with depression identified as an independent correlate in some cross-sectional studies [10,28,30–31]. Psychological distress may amplify pain perception and interfere with sleep initiation or maintenance, while persistent sleep disruption may further worsen emotional well-being [28,30–32]. Because most available evidence is observational, these findings suggest a potentially reciprocal association but do not establish causality [28,30–32].

3.3. Clinical Implications

The clinical relevance of sleep disturbances in ankylosing spondylitis is evident across multiple patient-reported outcomes. Poor sleep has been associated with higher disease activity, greater pain, impaired physical function, more severe fatigue, and poorer health-related quality of life [11,28,30–33]. Importantly, sleep quality has been shown to remain associated with disease activity even after adjustment for demographic and psychological factors, suggesting that it provides clinically meaningful information beyond conventional assessment [10]. Because disease activity and functional status in axial spondyloarthritis rely heavily on patient-reported measures, unrecognised sleep impairment may complicate the interpretation of these outcomes [7,10].

4. Fatigue

4.1. Prevalence and Characteristics

Despite considerable advances in the treatment of ankylosing spondylitis (AS), fatigue remains a common and burdensome symptom [9,34]. The reported prevalence varies across studies, but a recent systematic review and meta-analysis estimated that approximately one-half of patients with axial spondyloarthritis experience clinically significant fatigue [9].

Fatigue is heterogeneous in severity and clinical presentation, which complicates its assessment. Although the fatigue item included in the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) is widely used in clinical practice and research, it reflects only a single aspect of the symptom. More comprehensive patient-reported outcome measures are available, although their measurement properties and clinical applicability vary considerably [35,36].

4.2. Mechanisms and Contributing Factors

Fatigue may remain clinically relevant even in patients who meet remission or low-disease-activity criteria, suggesting that mechanisms beyond measurable disease activity may be involved [15,16]. Biological, clinical, and psychosocial factors are recognised to contribute to fatigue severity [9,11,32].

Inflammatory signalling may contribute to fatigue through its effects on central nervous system function [25,37]. In AS, tumour necrosis factor- α (TNF- α) and interleukin (IL)-17 are key mediators of musculoskeletal inflammation [2,12,13]. Evidence from broader inflammatory settings indicates that cytokine-mediated immune-to-brain signalling can induce sickness behaviour, characterised by fatigue, reduced motivation, cognitive changes, decreased physical activity, and social withdrawal [25,37]. Although this response is considered adaptive during acute inflammation, persistent immune activation may contribute to chronic fatigue; however, the specific relevance of this mechanism to AS remains incompletely established [25,37].

Individual studies have associated higher disease activity, persistent pain, morning stiffness, and poorer physical function with greater fatigue [11,38]. However, pooled evidence for disease activity and functional status has been inconsistent, reflecting substantial heterogeneity across studies [9].

Sleep-related and psychological factors also play an important role in the clinical expression of fatigue. Poor sleep quality is associated with greater fatigue severity in patients with AS [10,11,17]. Anxiety, depression, and psychosocial distress are likewise associated with greater fatigue and a heavier overall symptom burden [11,32].

4.3. Clinical Implications

For many patients with ankylosing spondylitis (AS), fatigue imposes persistent limitations across multiple areas of life. Greater fatigue severity is associated with poorer functional status, as reflected by higher Bath Ankylosing Spondylitis Functional Index (BASFI) scores [11,38]. It is also linked to poorer mental well-being, reduced participation in leisure activities, and difficulties in maintaining social relationships [32]. Together, these consequences contribute to poorer health-related quality of life [9,11,32].

Its impact is also evident in occupational functioning. In etanercept-treated patients with AS, fatigue independently predicted greater presenteeism and activity impairment, but not absenteeism or overall work productivity loss [39]. These findings indicate that fatigue may affect selected dimensions of occupational and daily functioning [39].

Given its broad effects on daily functioning and quality of life, fatigue warrants direct assessment as a distinct patient-reported domain [7,36]. Accordingly, validated patient-reported outcome measures are important for evaluating this symptom in clinical research and routine practice [7,36]. A systematic review identified multi-item instruments, including the Multidimensional Fatigue Inventory and Functional Assessment of Chronic Illness Therapy–Fatigue, among the most comprehensive measures available for axial spondyloarthritis. However, it also highlighted limitations in the quality and completeness of the psychometric evidence supporting existing instruments [36].

A more recent psychometric validation study conducted across the full spectrum of axial spondyloarthritis demonstrated that FACIT-Fatigue has good internal consistency, adequate convergent and known-groups validity, and sensitivity to change over time [40]. The study also established thresholds for classifying symptom severity and identifying meaningful within-patient improvement, supporting the use of this instrument as a fit-for-purpose measure in axSpA clinical trials and facilitating interpretation of changes in fatigue scores [40].

5. Clinical Management

5.1. Effects of Anti-inflammatory Treatment

Anti-inflammatory treatment remains central to the management of active axial spondyloarthritis [6]. Tumour necrosis factor inhibitors and interleukin-17 inhibitors can reduce fatigue, although comparative evidence does not establish the superiority of either drug class and most available data concern short- and medium-term outcomes [34]. TNF inhibition may also improve patient-reported sleep problems and fatigue [41,42], whereas objective polysomnographic changes remain inconsistent [23,27].

Ixekizumab, an IL-17A inhibitor, has been associated with improved patient-reported sleep outcomes, while secukinumab, another IL-17A inhibitor, has shown sustained reductions in fatigue [43,44]. These changes generally occur alongside improvements in pain, disease activity, physical function, and health-related quality of life [34,42–44]. Improvements in sleep and fatigue should therefore be interpreted as components of the broader clinical response rather than as clearly independent, symptom-specific effects.

5.2. Non-pharmacological Approaches

Non-pharmacological management should complement pharmacological treatment in axial spondyloarthritis. Patient education, regular exercise, and individually selected physiotherapy are recommended according to physical function, clinical status, and patient needs [6,45]. Exercise therapy can improve fatigue, pain, disease activity, physical function, spinal mobility, and aerobic capacity [45–47]. These benefits do not necessarily correspond to reductions in C-reactive protein or erythrocyte sedimentation rate, indicating that exercise may improve symptoms and functioning without consistently modifying conventional inflammatory markers [46]. The optimal type, intensity, frequency, and duration of exercise for specifically reducing fatigue remain uncertain; therefore, programmes should be adapted to individual capacity and maintained as part of long-term care [6,45,46].

Evidence for interventions specifically targeting sleep disturbances in AS is limited. When chronic insomnia is present, cognitive-behavioural therapy for insomnia (CBT-I) is recommended as first-line treatment, including in adults with medical comorbidities [48]. Sleep hygiene education may support treatment but should not replace structured CBT-I [48]. No disease-specific CBT-I trials were identified in the present narrative search; therefore, its use in patients with AS is currently extrapolated from general insomnia guidelines [48].

Discussion

Persistent fatigue or sleep disturbance despite improvement in disease activity should prompt a broader clinical assessment. In addition to possible residual inflammatory activity, clinicians should consider pain, physical limitations, psychological distress, chronic insomnia, obstructive sleep apnoea, and restless legs syndrome [6,10,15–17,19–22,29,32].

Available treatment studies commonly assess sleep and fatigue alongside broader measures of disease activity, pain, physical function, and quality of life [34,41–44].

Future studies should use standardised instruments and clinically meaningful response thresholds. Sleep and fatigue should also be included more often as prespecified or primary outcomes. Longitudinal research is needed to clarify how inflammation, pain, psychological distress, sleep impairment, and fatigue influence one another over time. In studies of sleep, combining patient-reported measures with objective assessment may also help explain why subjective improvement does not always correspond to changes in polysomnographic findings [23,27].

Conclusions

Sleep disturbances and fatigue are common and clinically relevant manifestations of ankylosing spondylitis that are associated with impaired physical functioning, psychological well-being, social participation, and health-related quality of life. Fatigue may additionally affect selected dimensions of occupational functioning. Although both symptoms are associated with pain and disease activity, neither can be explained by inflammation alone.

Their severity reflects interacting inflammatory, functional, sleep-related, and psychosocial factors. Anti-inflammatory treatment may improve fatigue and patient-reported sleep outcomes, but residual symptoms can persist despite adequate disease control. Their assessment should therefore consider both residual inflammatory activity and potentially modifiable non-inflammatory contributors.

Further longitudinal and intervention studies using standardised outcome measures are needed to clarify causal relationships and distinguish symptom-specific treatment effects from broader improvements in disease control. Better recognition of sleep disturbances and fatigue may support a more patient-centred evaluation of disease burden in ankylosing spondylitis.

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