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NEUROINFLAMMATION AND COGNITIVE DYSFUNCTION IN AXIAL SPONDYLOARTHRITIS

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Abstract
Axial spondylitis (AxSpA) is traditionally considered a chronic inflammatory disease primarily affecting the axial skeleton. However, in recent years, its extraskeletal manifestations, including nervous system involvement in the form of cognitive impairment and neuroinflammation, have attracted the attention of researchers. In AxSpA , these manifestations are underpinned by a multifactorial pathogenesis: systemic and neuroinflammation, chronic pain, sleep disturbance, comorbid depression and anxiety, and the impact of drug therapy. The aim of this review is to summarize current data on the prevalence, pathogenesis, clinical significance of cognitive impairment, and the role of neuroinflammation in AxSpA.

Keywords: Axial spondyloarthritis, neuroinflammation, cognitive impairment.

Introduction

Axial spondyloarthritis is a heterogeneous group of diseases characterized by chronic inflammation, leading to significant functional limitations and decreased quality of life. Genetic predisposition, particularly HLA-B27 status, plays a key role in the pathogenesis of the disease [1, 22, 28].

In 2009, the international society ASAS (The Assessment of SpondyloArthritis International The Society) proposed an expanded terminology for axial spondyloarthritis. In addition to classic ankylosing spondylitis (AS), this group included non-radiographic axial spondyloarthritis, psoriatic arthritis, spondyloarthritis associated with Crohn's disease and ulcerative colitis, and Reiter's disease [2, 6, 23, 28].

Ankylosing spondylitis (AS) is a chronic, progressive inflammatory disease associated with damage to the entheses and peripheral joints. The disease progresses through bone proliferation, ultimately leading to ankylosis of the spine and joints [6, 12, 26].

The disease has a demographic nature, with an average global prevalence of 0.1-1.5%. It has gender differences and is more common in men, with onset typically occurring before age 40. Unfortunately, statistics show that women experience a more severe course of the disease. The disease is socially significant, leading to early disability in the younger population [12, 31, 35].

Carriage of the human leukocyte antigen (HLA - B27) allele is a significant factor in the development of AS. HLA - B27 , which is associated with the MHC - I surface protein encoded by the MHC gene on chromosome 6, is the most important gene predisposing a person to AS. According to various authors, it is present in 65% to 100% of all patients with AS. However, its

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presence does not necessarily prove the presence of AS; it can also be found in healthy individuals and in other autoimmune diseases. The prevalence of HLA - B27 carriage in healthy individuals is demographically determined. According to international literature, it occurs in 4% to 50% of the healthy population, and only 0.07-10% of people (on average, 5%) develop AS. However, it should be noted that the risk increases in individuals with a family history of AS. The HLA - B 27 allele has more than 160 subtypes, but since only a few of them are widespread, tracing the association of a specific subtype with the incidence of AS presents certain difficulties [1, 3, 14, 19, 31].

Extra-axial manifestations include peripheral arthritis, enthesitis , and dactylitis. Among the extra-skeletal manifestations of axial spondyloarthritis, the most common are eye damage (uveitis), psoriasis, inflammatory bowel disease (Crohn's disease, ulcerative colitis), IgA nephropathy, aortitis, damage to the nervous system, etc. [14, 35].

Although the primary focus has traditionally been on joint and spinal involvement, in recent years there has been an increasing number of studies devoted to extra-axial manifestations, particularly nervous system involvement, particularly **cognitive impairment (CI) and neuroinflammation .** Compared to rheumatoid arthritis and systemic lupus erythematosus, neurocognitive aspects in patients with AxSpA have been less studied, leading to growing interest in the neurocognitive aspects of this pathology in recent years. Although the disease has not traditionally been associated with central nervous system involvement, accumulated data demonstrate impairments in memory, attention, cognitive flexibility, information processing speed, and executive functions in patients with AxSpA . These impairments have a direct impact on quality of life, professional and social adaptation. The severity of cognitive impairment varies and does not always correlate with the activity of the inflammatory process, which emphasizes the complexity of the interaction of immune, neurobiological and psychosocial factors [5,10, 11, 15].

Cognitive impairments, such as memory, attention, and executive function decline, are observed in a significant proportion of patients with AxSpA . Despite the absence of overt neurological disease, cognitive deficits can significantly impair daily functioning and quality of life. The frequency and severity of cognitive impairment in patients with AxSpA vary significantly across studies. However, several clinical studies and cross-sectional studies indicate that 25–50% of patients with AxSpA exhibit some manifestations of cognitive dysfunction (including mild and subclinical forms) when using sensitive instruments (MoCA , a battery of neuropsychological tests). When compared with matched controls, statistically significant delays in verbal memory, attention, and executive functions are observed. Meta-analyses of immune -mediated inflammatory diseases (IMIDs) show that cognitive impairment in systemic autoimmune inflammatory diseases is common and persistent. [5, 11]

Based on these data, it can be argued that cognitive impairment is an important, yet often ignored, aspect of AcSpA . Moreover, the severity of cognitive impairment may correlate with the level of inflammatory activity, indicating a role for inflammation in the development of these disorders. Cognitive impairment in AcSpA has a multifactorial pathogenesis, including systemic inflammation, neuroinflammatory mechanisms, chronic pain and its neuronal consequences, sleep disturbance, psychoemotional comorbidities , and the impact of therapy [15, 29].

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One of the leading factors contributing to cognitive impairment in axSpA is chronic inflammation. Chronic inflammatory activity in axSpA is accompanied by increased levels of proinflammatory cytokines (tumor necrosis factor-alpha, interleukins 17, -6, -1β), which can have adverse effects on the central nervous system (CNS): increasing blood-brain barrier permeability, activating microglia , disrupting synaptic transmission and neuroplasticity , particularly in the hippocampus and prefrontal cortex—structures critical for memory and executive function—causing neuroinflammation in the brain. A link between systemic inflammatory markers and cognitive decline has been demonstrated in a number of population-based and cohort studies of axSpA. These mechanisms are indirectly supported by neuroradiological and experimental studies, where inflammation is associated with changes in gray matter volume and functional connectivity [11, 29, 32]

Chronic inflammation can also lead to altered neuroplasticity and impaired neurotrophic support. Inflammatory cytokines can reduce levels of neurotrophic factors such as BDNF (brain - derived neurotrophic factor), which are necessary for the normal functioning of neurons and synaptic transmission, which in turn can lead to deterioration of cognitive functions [5, 14, 30].

Chronic pain, characteristic of AcSpA , is also a significant factor contributing to cognitive impairment. Pain impacts sleep quality, leading to impairments in memory, attention, and psychomotor function. Furthermore, pain can exacerbate depression and anxiety, which are themselves independent risk factors for the development of cognitive impairment. One study showed that AcSpA patients suffering from chronic pain exhibited decreased cognitive abilities, as evidenced by tests of attention, memory, and executive function. Clinical observations show a correlation between pain indices and a reduction in subjective and objective cognitive impairment; pain reduction is often accompanied by restoration of cognitive abilities [15].

Sleep disturbances (insomnia , sleep fragmentation , intermittent wakefulness) are common among patients with AcSpA and correlate with fatigue, depression, and cognitive decline. Chronic sleep deprivation impairs memory registration processes, attention, and reaction time; in patients with AcSpA, sleep correction is associated with improvements in a number of cognitive and functional indicators [5].

Depression and anxiety are common comorbidities in AxSpA . They themselves lead to a deterioration in cognitive speed, attention, and memory; the presence of depression increases subjective complaints of "cognitive decline" and worsens objective test performance. Therefore, depressive disorders in patients with AS not only worsen quality of life but may also mediate the effect of systemic inflammation on cognitive function [20].

Ankylosing spondylitis is traditionally understood as a musculoskeletal disorder of immune-inflammatory origin. However, the systemic nature of the disease, prolonged inflammatory activity, and severe chronic pain create a context in which the central nervous system (CNS) undergoes both functional (changes in network activity, fatigue symptoms, sleep disturbances) and structural changes. In addition to classic nonspecific symptoms (fatigue, decreased quality of life), patients with AS report cognitive and psychological impairments, which in some cases correlate with inflammatory markers and disease activity. This makes neuroinflammation a clinically and scientifically significant area of research in this pathology [13, 32].

Neuroinflammation is a local immune response of the central nervous system (CNS) characterized by activation of microglia and astrocytes , cytokine production, changes in BBB permeability, and

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involvement of pericyte and endothelial mechanisms [10]. Neuroinflammation is an important pathogenetic mechanism that links systemic inflammation in AxSpA with CNS dysfunction. Cytokines and other inflammatory mediators , such as activated macrophages and microglia , can penetrate the brain, where they cause gliosis —activation of glial cells (microglia and astrocytes), which contributes to neuronal damage and cognitive impairment [10, 16].

AxSpA is characterized by activation of the interleukin-23/interleukin-17 (IL-23/IL-17) axis, high production of TNF-α, and increased IL-6. These cytokines, circulating in the peripheral blood, can affect the CNS through several pathways: penetration through areas with relative loss of the BBB, as well as interaction with endothelial receptors and subsequent amplification of inflammatory signaling; signaling through peripheral nociceptors and vagal afferentation. IL-17A directly activates microglia and astrocytes , enhancing the synthesis of proinflammatory mediators, and TNF-α regulates the microglial phenotype and can mediate disturbances in synaptic plasticity. Together, these mechanisms are capable of initiating and maintaining neuroinflammation during long-term systemic activity of AxSpA [24, 34].

Chronic systemic inflammation contributes to the disruption of the BBB integrity: cytokines, free radicals, and endothelial dysfunction increase barrier permeability, facilitating the influx of peripheral immune mediators and cells into the brain parenchyma. This leads to increased microglial activation and local production of proinflammatory cytokines, which perpetuate a self-reinforcing inflammatory circle. In patients with AcSpA, direct evidence of persistent BBB dysfunction is still limited, but experimental logic and analytical data on related IOIs allow us to consider this mechanism as probable [10]. Microglia act as the first “front” of neuroinflammation : when activated, they produce TNF- α , IL-1β , IL-6, NO , and ROS , which affects neuronal networks and synaptic functions. Astrocytes , in turn, regulate homeostasis and maintain the BBB; With chronic stimulation, they acquire a reactive phenotype, contributing to neuronal dysfunction and metabolic support disorders. The dynamics of these cellular reactions under conditions of persistent systemic inflammation determine the scale and clinical manifestations of neuroinflammatory changes [10].

Peripheral inflammatory mediators can transmit signals to the brain via afferent fibers (including the vagus nerve), the spinal cord conduction system, and peripheral ganglia. Chronic nociceptive stimulation in AcSpA strongly involves spinothalamic and limbic structures, altering their functional relationships and potentiating neuroinflammation . Thus, the "pain phenomenon" and neuroinflammation are closely intertwined and mutually reinforce each other [2, 3, 4, 29].

Neuroinflammation manifests as a spectrum of symptoms ranging from nonspecific (fatigue, fogginess, decreased concentration) to pronounced neuropsychiatric changes (depression, anxiety) and neurocognitive dysfunction (decreased memory, attention, and executive function). Sleep disturbances and increased fatigue are also observed and often correlate with disease activity. Some reports have described isolated cases of demyelinating syndromes during biologic therapy , highlighting the need for vigilance in monitoring neurological events in patients on biologic therapy [17, 27, 33].

TNF-α blockade reduces systemic inflammation and has been associated with improvements in fatigue, depression, and some cognitive parameters in patients with immune-mediated inflammatory diseases in several studies. However, anti-TNF -α drugs are also associated with rare neurological side effects, including demyelinating events and exacerbation of pre-existing disorders. Thus, anti-

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TNF-α therapy potentially reduces neuroinflammation in most patients, but individualized monitoring is necessary due to rare neurological complications [17, 18, 21].

Since IL -17 plays a key role in the pathogenesis of AxSpA and can activate CNS cells (microglia , astrocytes), blockade of this axis should theoretically reduce neuroinflammation . Clinical studies confirm the effectiveness of IL-17 inhibitors in controlling systemic inflammation in AxSpA , data on direct neuroprotection or The neuroimaging effects are still limited, but molecular evidence supports the potential benefit of neuroinflammatory markers [7, 8, 24, 34].

Neuroinflammation is accompanied by clinical phenomena that significantly reduce quality of life: fatigue, sleep disturbances, decreased cognitive performance, depression, and decreased work capacity. These manifestations are often underestimated in routine rheumatology practice, leading to an incomplete assessment of the disease burden and limiting opportunities for therapy optimization. Integration of neurocognitive assessment into clinical management protocols for patients with AxSpA appears warranted and potentially improves treatment outcomes [13].

Elevated levels of cytokines (e.g., TNF - α , IL -6, and IL-17) can enhance neurodegeneration and modify synaptic plasticity, which impairs cognitive processes such as memory and attention. Cytokines can also increase the permeability of the blood-brain barrier, leading to additional penetration of inflammatory molecules into the brain and increasing the level of neuroinflammation [10].

Magnetic resonance imaging (MRI) of the brain has shown that patients with AS may exhibit changes in brain structures such as the hippocampus and frontal lobes, which are responsible for memory, attention, and executive functions. Patients with high levels of inflammation in the body also showed signs of volumetric changes in these areas, which may explain the decline in cognitive abilities. Research suggests that the hippocampus, which plays a key role in long-term memory formation, may undergo structural changes due to neuroinflammation , leading to cognitive impairment [9, 10, 11].

Further study of nervous system involvement requires large prospective studies including standardized neuropsychological testing in patients with AxSpA , biomarker measurements neuroinflammation and cytokines, neuroimaging (MRI/MSCT), as well as randomized studies assessing the impact of different treatment regimens (anti-TNF-alpha, anti-IL-17) on cognitive dynamics.

Thus, the study of the neurological aspects of axial spondyloarthritis is an important area of modern clinical neurology and rheumatology, having both diagnostic and prognostic significance.

Conclusion

Axial spondyloarthritis is a serious systemic disease that requires a multidisciplinary approach. Neurological complications associated with AsSpA can manifest as a variety of clinical symptoms, depending on the location of the inflammatory process, the degree of spinal ossification, and vascular involvement. Timely recognition of these manifestations is crucial for preventing disability and selecting the optimal treatment strategy.

Cognitive impairment in ankylosing spondylitis is a clinically significant problem caused by the complex interaction of systemic and neuroinflammation , chronic nociceptive stimulation, sleep disturbance, and psychoemotional factors. This necessitates systemic screening of cognitive function in patients with AS, particularly those with risk factors, and the use of multilevel interventions—from

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optimizing anti-inflammatory therapy to sleep modification, pain management, and psychotherapy. Only the integration of rheumatological, neurological, and psychiatric approaches will improve the cognitive prognosis and quality of life in these patients. Further prospective studies assessing neuropsychological and biomarker outcomes are a priority.

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