

NEUROPATHIC COMPONENT OF PAIN IN RHEUMATOID ARTHRITIS AND TREATMENT METHODS

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Abstract:

The article discusses the pathogenesis of pain in rheumatoid arthritis (RA), presenting a large body of literature and findings from our own research on the prevalence and significance of polyneuropathy in the development of peripheral neuropathic pain. In RA, peripheral nervous system damage—such as sensory or sensorimotor axonal neuropathy, demyelinating polyneuropathy, and myelopathy—has been documented. Joint deformities, limitations in active and passive movements, and muscle atrophy make it challenging to identify the exact cause of paresis, whether it stems from joint pathology or peripheral nerve damage. Comprehensive treatment of the neuropathic pain component in RA includes nonsteroidal anti-inflammatory drugs, glucocorticosteroids, biologic therapies targeting the underlying disease, and centrally acting medications. B vitamins play an essential role in restoring affected neurons, regenerating peripheral nerves, and aiding myelin sheath synthesis, offering both regenerative and analgesic effects.

Keywords: rheumatoid arthritis, polyneuropathy, B vitamins, pain syndrome.

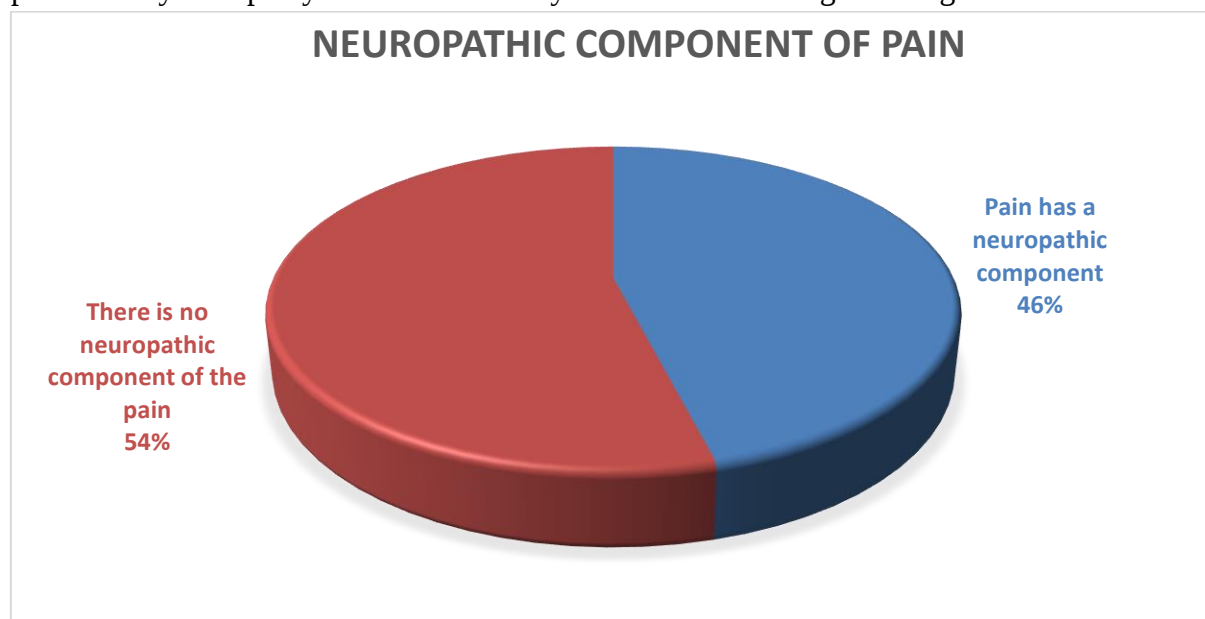
Introduction

Rheumatoid arthritis (RA) is a chronic systemic connective tissue disease that primarily affects peripheral (synovial) joints, leading to progressive damage, erosive-destructive polyarthritis, multi-organ involvement, and severe complications such as secondary amyloidosis. Currently, RA affects approximately 0.5-1% of the global population, with over 17.6 million people living with the condition. RA is more common in women, occurring 2-3 times more frequently than in men. It accounts for up to 10% of joint diseases, with an annual incidence rate of 0.02%. Research indicates that genetic factors influence the extent of ankylosis, deformity, and contractures in joint structures associated with RA. The most characteristic feature of RA is chronic pain, accompanied by progressive joint damage, reduced mobility, fatigue, and extra-articular manifestations, typically concentrated around the joints. This chronic pain syndrome is the primary complaint among patients and significantly reduces quality of life. Pain

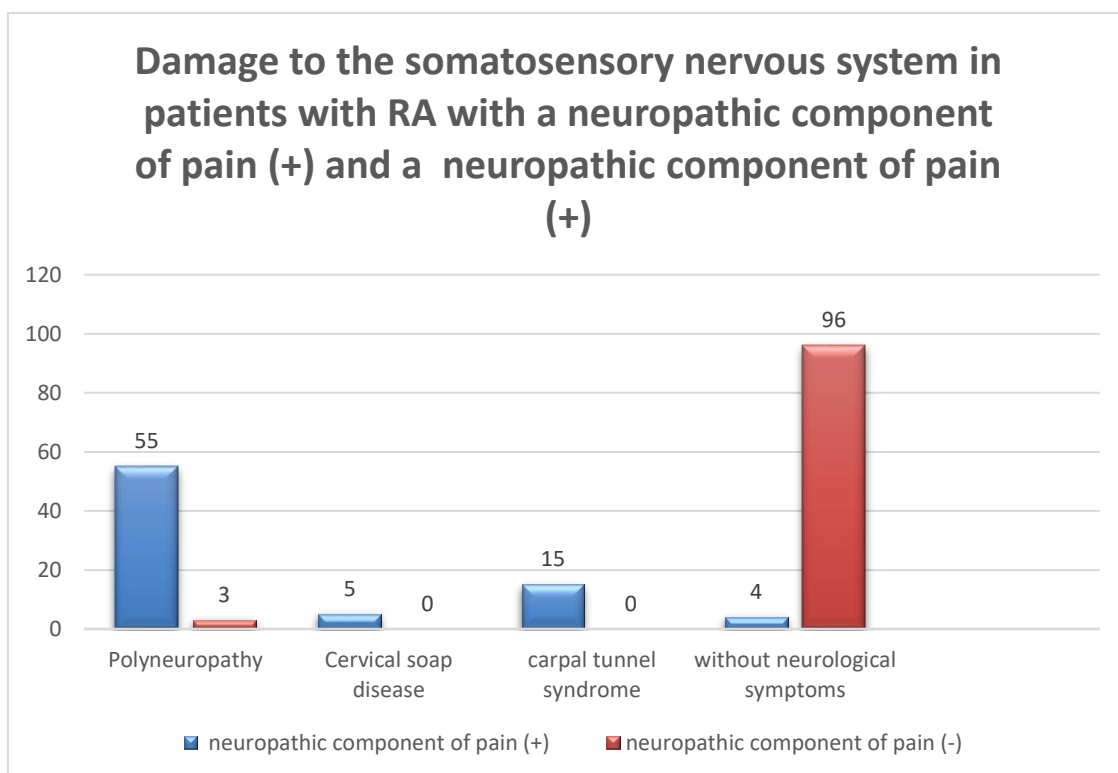
serves as an indicator of disease activity; however, disease activity does not always correlate with pain intensity or joint dysfunction.

Neurological Changes in Rheumatoid Arthritis

Numerous clinical and experimental studies highlight the role of neurogenic mechanisms in RA pain pathogenesis. Alongside the clinical signs of inflammation, patients with RA experience specific sensory symptoms typical of neuropathic pain. Peripheral nervous system damage is a common neurological issue in RA. A study by American rheumatologists on the clinical, electrophysiological, and pathological characteristics of peripheral nervous system damage in 108 RA patients found electrophysiological signs of neuropathy in 62 subjects (57.4%). Of these, 53 patients (85.5%) had pure sensory or sensorimotor axonal neuropathy, while 9 (14.5%) exhibited demyelinating polyneuropathy. Carpal tunnel syndrome was identified in 11 of the 108 patients. Peripheral nervous system damage presented various clinical symptoms, including pain, paresthesia, and motor and sensory impairments. The authors noted that these symptoms might mimic or accompany joint pain. However, no correlation was found between neurological manifestations and factors such as disease duration, presence of erosions, joint deformities, or use of anti-inflammatory drugs. According to the literature, the frequency of neurological complications in patients with RA varies widely, ranging from 0.5% to 85%. These complications are primarily polyneuropathies (sensory, motor, or sensorimotor) and various mononeuropathies, with cervical myelopathy being significant. Polyneuropathy affecting both motor and sensory fibers is observed in more than 50% of cases. Diagnosing motor disorders remains challenging, as existing joint deformities, which limit active and passive movements and are often accompanied by muscle atrophy, can obscure the causes of paresis. Polyneuropathy is more commonly associated with long-standing disease.



In 2023, we conducted a study to identify the neuropathic component of pain in patients with RA. We examined 183 patients with confirmed RA (mean age 48.5 ± 11.7 years) and a disease duration ranging from 6 months to 35 years (mean duration 12.1 ± 7.6 years). Based on the DN4 questionnaire used to determine pain type, 73 patients (46%) scored 4 or more points, indicating a neuropathic nature of the pain. Statistical analysis of two groups of patients (with neuropathic and non-neuropathic pain components) revealed that increased age, longer disease duration, higher clinical stage, reduced functional capacity, and greater pain levels were associated with a higher likelihood of neuropathic pain components. Neurological examination in 95% of patients with a neuropathic component of pain showed evidence of peripheral nervous system involvement, including distal sensorimotor polyneuropathy (56%), carpal tunnel syndrome (15%), mononeuropathy (19%), cervical myelopathy (5%), and combined polyneuropathy with tunnel syndrome (4%). Polyneuropathy was more common among elderly patients with long-term RA, supporting findings from international studies.



Our study showed that the chronic joint pain syndrome in RA has a mixed nature. In addition to the nociceptive component caused by inflammation, neuropathic component of pain prevails in 43% of patients, which requires complex therapy. In addition to the main anti-inflammatory, glucocorticosteroid and genetic engineering biological therapy directed at the underlying disease and nociceptive component, therapy for the neuropathic component should be considered. According to international

recommendations, in addition to the above drugs, centrally acting drugs (anticonvulsants and antidepressants) and B vitamins should be used to treat the neuropathic component of pain. For many years, scientists have been using B vitamins in various neurological diseases, primarily vitamins B1 (thiamine), B6 (pyridoxine) and B12 (cyanocobalamin), which have a unique mechanism of action. conducted clinical research.

- Thiamine (B1) has a reparative effect on affected neurons and also slows down the progression of vascular damage.
- Pyridoxine (B6) has a neurotropic effect (activates the synthesis of transport proteins in the myelin sheath of nerve fibers and axons, accelerates the regeneration process of peripheral nerves) and has an analgesic effect.
- Cyanocobalamin (B12) has an active effect on cell energy supply, protein synthesis and regeneration of nerve tissue.

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