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THE ROLE OF DIET AND PHYSICAL ACTIVITY IN RHEUMATOID ARTHRITIS TREATMENT – A REVIEW

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ABSTRACT

Rheumatoid arthritis (RA) is a chronic, multifactorial autoimmune disease characterized by joint inflammation, systemic complications, and progressive disability. Pharmacological therapies such as disease modifying management (DMARDs) and symptomatic treatment (NSAIDs and GCs) remain the cornerstone of RA management, increasing evidence highlights the significant role of non-pharmacological strategies in enhancing the treatment outcomes. This review explores the impact of physical activity and diet strategies on RA progression and control. Physical activity, particularly resistance and aerobic training, improves muscle mass, joint function, and cardiovascular health, counteracting RA-related cachexia and reducing systemic inflammation. Dietary interventions, especially Mediterranean and vegan diets, are reported to have anti-inflammatory benefits due to high content of antioxidants, omega-3 fatty acids, polyphenols, and phytochemicals. Specific nutrients like vitamin D, olive oil, and polyphenol-rich foods exhibit immunomodulatory and anti-osteoclastic effects. Integrating dietary, lifestyle, and pharmacology therapies may lead to disease remission, reduce treatment resistance, and improve patients' quality of life.

KEYWORDS

Diet, Mediterranean Diet, Physical Activity, Rheumatoid Arthritis, Treatment

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Introduction

Rheumatoid arthritis (RA) is a multifactorial autoimmune disease of unknown ethology which occurs in about 5 per 1000 people all over the world. Primarily it's affecting the joints, then extra-articular manifestations can appear. Due to chronic inflammatory process appears damage of joints as well as extra-articular organs like heart, kidney, lung, eye, digestive system, skin or nervous system [1,2]. It can affect people of any age, the peak occurs at age 50-59 years old, and is 2-3 times more frequently between women than men [3]. RA while affecting joints, causes pain, swelling, stiffness, and potential joint destruction. The inflammation process can lead to the degeneration of cartilage and bone, affect joint function and lead to deformities [4]. It usually begins with the smaller joints, like the hands and feet, but can progress to involve larger joints – knees, shoulders, elbows. Due to systemic inflammation with joint problems semi-occur fatigue, fever, general feeling of unwellness [5]. The pathogenesis of RA is connected with a complex interplay of genetic and environmental factors. Genetic susceptibility plays an important role, due to the presence of the HLA-DRB1 gene [6]. Other genetic markers of severity such as TRAF1, PSORS1C1 and microRNA 146a are associated as well with joints damage [7]. Environmental factors, such as infections, smoking, can activate the immune system in genetically predisposed individuals, contributing to the higher production of autoantibodies like rheumatoid factor (RF) and anti-citrullinated protein antibodies (ACPA) [8]. Family history of RA, other immune diseases, poor dental health, viral infections can also increase the likelihood of developing RA [3].

Treating of RA is concentrated on reducing inflammation, preventing joint damage, and improving quality of life. The pharmacological strategies include Disease-Modifying Antirheumatic Drugs (DMARDs) like methotrexate, tumour necrosis factor (TNF) inhibitors, interleukin-6 (IL-6) inhibitors, and Janus kinase (JAK) inhibitors, as well as glucocorticoids (GCs) with nonsteroidal anti-inflammatory drugs (NSAIDs) like naproxen, ibuprofen or coxibs [9, 10, 11, 12]. However there are proofs of positive effects of non-pharmacological ways of treatment RA which include physical activity. Even single exercises or other physical activity interventions can reduce the global impact of disease and improve the quality of life [13]. Diet rich in omega-3 polyunsaturated fatty acids, vitamin D supplementation, dietary sodium restriction, and Mediterranean diet were proven to reduce RA disease activity and lower failure rate of pharmacotherapy [14].

Non-pharmacological treatments of RA which include physical activity, healthy diet, changing habits can lead to improvement of joint function, reduce of pain, and enhance of the quality of life. Lifestyle modifications such as Mediterranean diet, quitting smoking, healthy weight can reduce the process of inflammation and help to maintain joint mobility [13,15].

Methods

A literature analysis was performed using the PubMed and Google Scholar database. It is based on secondary data from published meta-analyses, systematic review, and network meta-analyses. The keywords used were rheumatoid arthritis alone or in combination with pharmacological treatment, non-pharmacological treatment, dietary intervention, physical activity, quality of life. Only publications in English were used. Articles of various types were analysed. This narrative review synthesizes current research findings and official guidelines from leading health organizations, such as World Health Organization and the American College of Lifestyle Medicine.

Rheumatoid arthritis – criteria

The 2010 classification criteria for RA, developed by the American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR), assess various factors, including risk factors, the number and type of joints involved, and the duration of symptoms. This approach shifts the focus from managing late-stage disease to the early detection of RA [16]. The classification system highlights conditions linked to specific scores, which must be reassessed over time:

- 2-10 large joints – 1 score;
- 1-3 small joints – 2 score;
- 4-10 small joints – 3 score;
- >10 joints (≥1 small joint + others) – 5 score;
- Negative RF and ACPA – 0 score;
- Low-positive RF or/and ACPA ≤3× upper limit – 2 score;
- High-positive RF or/and ACPA >3x upper limit – 3 score;
- Abnormal erythrocyte sedimentation rate (ESR) or/and abnormal C-reactive protein (CRP) – 1 score;
- Normal level of CRP and ESR – 0 score;
- Patients with symptoms like: pain, swelling and tenderness ≥6 weeks – 1 score [12, 17].

Patients with the total score of 6 or more can be diagnosed with RA. There must be met two mandatory criteria to qualify for a new series of tests. First one is the presence of synovitis, evidenced by swelling in 1 or more joint, as assessed by a specialist. It should exclude the typical joints affected by osteoarthritis, like the first metatarsophalangeal joint or the distal interphalangeal joint. The second one is the fact that the patients has no alternative diagnosis for synovitis. What is more, large joints include elbows, shoulders, hips, knees, and ankles, the small joints refer to proximal interphalangeal joints, wrists, and the second through fifth metatarsophalangeal joints [18].

Diagnostic and imaging in RA

Due to diagnostic and prognostic role of biomarkers, they found their use in diagnosing RF. The latest classification includes four biomarkers – RF, ACPA, ESR and CRP [19]. However there have been found other diagnostic proteins which are useful in early diagnosis of RA, like antibodies against mutated citrullinated vimentin (anti-MCV), antibodies against carbamylated proteins (anti-CarP) and 14-3-3 eta protein [20]. Predictive function of biomarkers plays a significant role in the therapeutic management and establishment of effective treatment in RA [21].

The ACR-EULAR 2010 classification includes ultrasonography, computer technology (CT) and magnetic resonance imaging (MRI) as tools in imaging and establishing an early diagnosis of RA, which is crucial for effective management and prevention of irreversible joint damage [22].

Ultrasonography in a non-invasive technique has an ability to detect early synovial inflammation and joint effusions. Due to identifying of synovial membrane thickening, increased vascularization, and early erosive changes, it is one of first techniques used to imagine visible changes in RA [23]. Doppler ultrasound has an ability to differentiate active from inactive inflammatory tissues [24]. CT technique is not common used for the fact of its ionizing radiation and limited soft tissues contrast, when MR is an accurate tool to detect an early stage of RA [25, 26]. MR provides detailed images of soft tissues, bones, allowing to the detection of synovitis, bone marrow edema, bone erosions [26].

Pharmacological treatment

The role of new therapeutic options in RA is reducing symptoms, preventing complications and slowing the progression of disease. According to EULAR recommendations there are two perspectives of pharmacological treatment: symptomatic treatment (NSAIDs and GCs) and disease modifying management (DMARDs) [27].

NSAIDs (naproxen, ibuprofen, coxibs) by inhibiting cyclooxygenase (COX), especially COX-2 which level is increased during inflammation, are commonly used during the acute phase of the disease to reduce pain [28]. Even 66% of RA patients regularly take NSAIDs, depending on disease activity, pain intensity, or functional impairment [29]. Glucocorticoids (prednisone, hydrocortisone, dexamethasone, prednisolone) due to their anti-inflammatory effect are as well often used to control the disease activity. Their effect increase in combination with disease-modifying antirheumatic drugs (DMARDs) by helping to control disease activity and inhibiting radiographic progression [30, 31]. However there are many long-term side effects of using GCs

like weight gain, water retention, diabetes, or bone thinning. For this reason they are recommended for only short-term use [31].

Disease-modifying antirheumatic drugs (DMARDs) are categorized into three main types: conventional synthetic DMARDs (csDMARDs), biologic DMARDs (bDMARDs), and targeted synthetic DMARDs (tsDMARDs). According to EULAR recommendations on RA, pharmacological therapy base on csDMARDs is supposed to be started right after having diagnosed with RA and methotrexate (MTX) should be in the first-line treatment strategy [32]. When csDMARDs are insufficient, bDMARDs like tumour necrosis factor (TNF) inhibitors (adalimumab, etanercept, golimumab), interleukin-6 (IL-6) inhibitors (tocilizumab), T-cell co-stimulation inhibitors (abatacept), B-cell depletors (rituximab), and IL-1 inhibitors (anakinra) are taken into consideration and tsDMARDs (tofacitinib, baricitinib) which offer oral treatment options [33]. Early initiation of DMARD therapy is crucial for achieving remission or low disease activity [34].

Non-pharmacological treatment

In 2021 EULAR Task Force invented the definition of difficult-to-treat rheumatoid arthritis (D2TRA) consisting of three major points: failure of at least 2 biological or targeted synthetic DMARDS, presence of active or progressive disease; and problematic management perceived by the rheumatologist or the patient. While advancements in pharmacological treatments continue,, D2TRA underlines the need for using additional therapeutic strategies in treatment of RA beyond medications alone. Particularly in D2TRA patients issues such as drug intolerance, limited work capacity, or persistent pain emphasize the value of integrating non-pharmacological strategies into treatment plans. Connecting pharmacological and non-pharmacological therapies have an additional, synergetic effect, enhancing overall treatment overcomes. Patients with established, active disease, as well as with long-lasting symptoms can be partially applied to D2TRA patients. Non-pharmacological treatments for RA include: exercise therapy, psychological interventions, physiotherapy and balneotherapy, and dietary interventions [35].

Physical activity

Patients with RA are reported to benefit from physical activity and occupational therapy. They are recommended to perform exercise regularly to maintain joint mobility and strengthen the muscles around the joint. Swimming, yoga, and thai chi are the examples of movement exercises that are less traumatic for the joints. Using heat and cold packs before and after exercise helps minimalize painful symptoms [36]. Lastly, researchers are studying different types of connective tissue collagen to better understand and reduce RA disease activity [37]. Patients with RA are reported to be less physically active and have aerobic capacities, 20-30% lower cardiorespiratory fitness than healthy patients. Exercise training and increased physical activity reduce cardiovascular events in general population. Meta-analyses of exercise-based cardiac rehabilitation estimate that mortality is reduced by around 20 to 30%. Since cardiovascular disease (CVD) is the main cause of reduced life expectancy among patients with rheumatoid arthritis, the likely cardioprotective benefits of exercise and regular activity for RA patients should not be overlooked [38]. Approximately two thirds of RA patients are noticed to suffer from cachexia defined as a loss of body cell mass with dominative skeletal muscle, stable bodyweight with the decrease in muscle mass and increase in fat mass [39]. It was proposed that cachexia in RA occurs due to the excess production of proinflammatory cytokines, especially TNF- α, which is catabolic and disrupts the balance between protein degradation and protein synthesis in RA [40]. High-intensity resistance exercise has been shown to safely reverse cachexia in RA patients, leading to improved muscle mass, reduced disability, and better physical function. For instance, a 24-wee progressive resistance training (PRT) program significantly increased lean body mass, reduced fat mass, and improved muscle strength and physical function. In contrast, low-intensity range exercises in a matched control group had no effect on body composition or function [38]. RA causes inflammation of tendon sheaths, leading to synovial hypertrophy and sometimes tissue infiltration into tendons which also lose the elasticity with age, inactivity, or disuse. Ligaments, another essential component of the joint, are similarly affected. Research shows that exercise strengthens ligaments, while even short-term immobilization weakens them. Therefore, regular physical activity is essential for RA patients to maintain healthy ligaments and overall joint function [41, 42].

Vitamin D

The link between vitamin D and RA is complex, as vitamin D plays a role in immune regulation relevant to RA pathophysiology. Deficiency is common among the patients, and the COMORA study, made across 15 countries, confirmed the low vitamin D levels regardless of geographic latitude. These low levels are associated with higher disease activity, greater corticosteroid use, and increased comorbidities. Despite this, vitamin D supplementation does not appear to modify disease activity. In a 12-wee randomized placebo-controlled trial (n=117), weekly doses of 50,000 IU of vitamin D had no effect on RA activity. Vitamin D's anti-inflammatory effects may steam from suppression of IL-17+ and IFN γ+ T cells. RA patients on steroid therapy may still require calcium and vitamin D to prevent osteoporosis, and possibly to help alleviate anxiety and depression symptoms [43, 44, 45].

Anti-Oxidants

Oxidative stress is significantly increased in RA, with elevated levels of reactive oxygen species, lipid and protein oxidation, DNA damage, and reduced antioxidant defences. However, evidence for the benefits of antioxidant supplementation in RA remains conflicting. One study in 40 women with RA showed that daily supplementation with selenium, zinc, and vitamins A, C, and E improved oxidative stress markers and disease activity, and did not reduce joint pain or swelling. Low serum selenium is strongly associated with RA and copper levels appear to correlate with disease activity [46,47].

Mediterranean Diet

Mediterranean diet is a diet rich in vegetables, fruits, whole grains, seeds, nuts, and olive oil as a main source of dietary fat. Consumption of low-fat dairy products, white meat, and eggs is moderated while reducing red meat, processed products, and sweets to a minimum [48]. It is rich in oleic acid, omega-3 fatty acids, unrefined carbohydrates, and phytochemicals. A study conducted by Sköldstam et al. found that a Cretan Mediterranean Diet reduced inflammation and improved vitality and physical function in RA patients. Olive oil has antioxidant and anti-inflammatory properties due to its high oleic acid content, which can be metabolized into compounds with effects similar to omega-3 fatty acids. In a study on CIA mice, Rosillo et al. showed that extra virgin olive oil reduced levels of COMP and MMP-3, markers of joint damage, and lowered expression of inflammatory cytokines like IL-1β, TNF-α, and IL-17. What is more, olive oil seems to suppress STAT-3 activation, which is involved in synovial cell proliferation and Th17 differentiation. It inhibits MAP (JNK, p38) and NF- κB signalling pathways, reduces pro-inflammatory gene expression, and potentially limits osteoclast-driven joint destruction. Mice fed with olive oil showed less joint swelling, cartilage damage, and arthritis progression, suggesting olive oil may help prevent RA [49, 50, 51, 52].

Vegan Diet

A vegan diet, which excludes all animal products, has been shown to support clinical improvement and even remission of RA patients. It may be connected with the reduction of immune reactivity to certain dietary antigens eliminated from the gut after dietary change. Fasting has also been associated with beneficial effects, Hafström et al. reported reductions in morning stiffness, ESR, articular index, acute-phase proteins – orosomucoid, C3, and haptoglobin, along with increased haemoglobin levels. Neutrophil lysozyme release, lined to joint inflammation mediator leukotriene B4 (LTB4) by neutrophils dropped significantly after fasting [53, 54]. During fasting, ketone bodies such as β-hydroxybutyrate (BHB) increase and act as alternative energy sources. BHB has been shown to inhibit nLRP3 inflammasome activation – responsible for releasing IL-1β and IL-18, two key pro-inflammatory cytokines in RA. The anti-inflammatory mechanism suggests that fasting and ketogenic diets could modulate inflammation via BHB-driven suppression of inflammasome pathways [55].

Polyphenols

Polyphenols, contained in fresh fruits, vegetables, and spices may have the beneficial anti-oxidative effects in RA patients. Dried plum, red grape skin, grapefruits, soybeans, black pepper, ginger, caraway, paprika, clove, wheat, oats, cocoa, and many more are reported to be rich in polyphenols. In a study on transgenic mice overexpressing TNF month-long supplementation with dried plum slowed evaluation of arthritis, reduced joints' bone erosion comparing to mice on normal diet. Dried plum rich also in neochlorogenic acid seems to inhibit the TNF-inducted osteoclast formation, reduce in the number of tartrate-resistant acid phosphatase (TRAP)-positive cells, which are involved in osteoclastogenesis [56, 57]. The anti-inflammatory potential of equol, a major metabolite of soy isoflavones, has been examined in mice with

collagen-inducted arthritis (CIA). Equol reduced arthritis severity and slowed bone mineral density loss. Its immunomodulatory effects included suppression of IL-6 and its receptor in inflamed tissues. It also influenced bone metabolism by downregulating genes linked to osteoclast activity, impaired bone formation, and cartilage degradation [58].

Conclusions

The integration of diet and physical activity into RA treatment offers a valuable adjunct to pharmacological therapy. Evidence supports that regular exercise, especially resistance and aerobic training, improves physical function, limits joint damage, and counters RA-associated comorbidities such as cachexia or cardiovascular disease. Diet rich in anti-inflammatory compound, like Mediterranean and vegan diets, can modulate immune response, reduce oxidative stress, and may slow the symptoms progression. Vitamin D, omega-3 fatty acids, and polyphenols play a specific role in regulating inflammation and bone metabolism. Personalized lifestyle interventions may enhance therapeutic efficacy and long-term disease management. Future research may clarify optimal protocols and exercise regimens tailored to individual RA phenotypes.

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