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LIFESTYLE MODIFICATIONS FOR MANAGING ANKYLOSING SPONDYLITIS: A CLINICAL STUDY.

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Abstract

Pain, stiffness, and a gradual reduction in spinal mobility are symptoms of Ankylosing Spondylitis, a chronic inflammatory rheumatic disease that mostly impacts the axial skeleton. Physical ability, personal satisfaction, and long-term health effects are all profoundly affected by the illness. Lifestyle changes have become an important supplementary method for managing disease progression and enhancing patient wellbeing, in addition to pharmaceutical therapy including biologic medicines and nonsteroidal anti-inflammatory medications. Lifestyle changes as a treatment for Ankylosing Spondylitis are the focus of this clinical trial. Interventions such as regular exercise, correcting posture, physiotherapy, balanced eating, quitting smoking, managing stress, and getting enough sleep are the main emphasis of the study. when a certain amount of time had passed, researchers tracked a group of individuals with spinal cord injuries to see how their pain levels, mobility, functional capacity, and quality of life changed when they started following a regimen of prescribed lifestyle adjustments. The effects of these changes were assessed using clinical measures and outcomes that patients themselves reported. The results show that physical activity, breathing exercises, and posture training are the most effective lifestyle modifications for reducing stiffness and discomfort and increasing mobility and functional ability. Physical endurance and inflammation management were both improved when people avoided smoking and ate healthily. These findings point to the need of lifestyle-based therapies in the overall therapy of Ankylosing Spondylitis, complementing medical treatment. Researchers found that individuals with Ankylosing Spondylitis had better disease control and a higher quality of life when lifestyle modification methods were included of their regular clinical treatment. We need more large-scale research to solidify our understanding of this chronic inflammatory illness and to create uniform guidelines for managing it through lifestyle changes.

Keywords: *Modifications, Ankylosing, Spondylitis, Managing*

Introduction

Pain, stiffness, and a gradual decrease in spinal mobility are symptoms of Ankylosing Spondylitis (AS), a chronic inflammatory rheumatic illness that mostly impacts the spine and sacroiliac joints. It is one of several spondyloarthropathies that cause inflammation in the axial skeleton and entheses (the places where ligaments and tendons connect to bone). Males are more likely than females to experience the onset of the disease in early adulthood. As the condition progresses, persistent inflammation can cause structural abnormalities such vertebral fusion, which in turn reduces flexibility and causes the stooped posture that is distinctive of advanced stages of the disease. Although the specific reason for ankylosing spondylitis is still not known, it is thought to be influenced by both hereditary and environmental

factors. The HLA-B27 gene is associated with the disorder in many people, making it one of the strongest genetic correlations. Asthma symptoms include persistent low back discomfort, stiffness upon waking, extreme weariness, and impaired mobility. Daily activities and general quality of life can be greatly diminished if the condition is not well treated. Pharmacological and non-pharmacological methods are usually used together in the management of ankylosing spondylitis. To alleviate inflammation and manage symptoms, doctors frequently recommend medications such biologic treatments, disease-modifying antirheumatic medicines (DMARDs), and nonsteroidal anti-inflammatory drugs (NSAIDs). Still, it's possible that short-term medical interventions won't be enough to forestall permanent functional decline. There has been a shift in focus toward lifestyle-based therapies as a means to augment traditional treatment and assist patients in preserving their autonomy and mobility. The long-term therapy of ankylosing spondylitis heavily relies on lifestyle adjustments. Spinal flexibility, better posture, and less stiffness can all be achieved by regular physical exercise and treatment. Preserving spinal mobility and improving respiratory function can be achieved by workouts including yoga, swimming, stretching, strengthening, and breathing exercises. On top of that, you may lessen the likelihood of spinal deformities and pain by utilizing ergonomic furniture, sleeping well, and keeping your posture straight while you go about your day. Inflammatory processes and general health in ankylosing spondylitis patients are impacted by dietary and nutritional factors as well. One way to promote immune function and lower systemic inflammation is to eat a balanced diet that is high in fruits, vegetables, whole grains, lean meats, and substances that reduce inflammation. In addition, there is evidence that healthy lifestyle choices, like not smoking, managing stress, and keeping a healthy weight, can enhance health outcomes and overall well-being. The increasing importance of lifestyle-based management measures in the treatment of ankylosing spondylitis is justified by the disease's chronicity and the lasting effects it has on physical functioning. Aiming to improve symptoms, functional ability, and overall wellbeing in patients diagnosed with ankylosing spondylitis, this present clinical investigation intends to investigate the effect of different lifestyle adjustments. Better disease control and improved patient outcomes can be achieved by incorporating lifestyle management into normal therapy procedures, and this study aims to do just that by analyzing the efficacy of various interventions.

Physical activity and sedentary behaviour

The PA spectrum primarily consists of sedentary behavior, PA, and scheduled activity. The general population is linked to a lower risk of CVD when they engage in higher PA and reduce their sedentary time (Wilmot et al., 2012). This is because exercise has anti-inflammatory effects (Beavers et al., 2010), improves vascular function (Di Francescomarino et al., 2009), and improves cardiometabolic health (Palmefors et al., 2014). Meeting the recommendation of at least 150 minutes of moderate to vigorous physical activity each week has been linked to a 35% lower risk of cardiovascular disease mortality, according to public health recommendations (UK CMO recommendations Writing Group, 2019; Wahid et al., 2016). Due to obstacles such as pain, exhaustion, immobility, and worries about pain and harm, patients with AS are more likely to be inactive than the overall population (Rasmussen et al., 2020; Liu et al., 2021). Because of this, it is commonly believed that AS patients are even more likely to be sedentary and less active than the general population, which increases their risk of cardiovascular disease.

The severity of AS can differ greatly from one patient to another and may be linked to persistent low-grade inflammation (Spoorenberg et al., 2005; Bakland et al., 2011). illness therapy for AS must prioritize increases in physical activity and inactive time due to the inflammatory character of the illness (Regel et al., 2017). Improved patient outcomes and reduced risk of co-morbidities like CVD can be achieved by investigating the advantages of increased PA and decreased sedentary time in individuals with AS. Coulter et al. (2020) found that patients with AS who engaged in increased physical activity and had reduced sedentary behavior had better function, exercise capacity, and quality of life. However, it is important to note that cross-sectional studies have limitations in their design and cannot establish a cause and effect relationship. Therefore, in order to learn how PA, exercise, and sedentary behavior affect health outcomes and inflammation in AS patients, high-quality randomized controlled trials and longitudinal research are required. It is crucial to study various modalities and forms of exercise since AS influences a wide range of structures and processes. Supplementary Table 1 shows that 45 papers were found in the present search that looked at the effects of physical activity and PA on disease activity in AS patients. Nevertheless, there has been a lack of research on the topic of reducing periods of inactivity. Given that AS patients spend a lot of time sitting (Coulter et al., 2020; O'Dwyer et al., 2015), as well as the correlations between inflammation (Elhakeem et al., 2018) and cardiovascular disease (CVD) (Wilmot et al., 2012), it would be prudent for future research to examine the impact of reducing sitting duration. Exercise was shown to significantly enhance the results in 27 out of 45 research (~82%) that examined BASDAI (Supplementary Table 1). Approximately 80% of the trials (33 out of 41) found that exercise significantly improved BASFI. As shown in Supplementary Table 1, this amounted to 19 out of 26 studies (~73%) pertaining to BASMI. Intervention intensity, frequency, program type, and length of time were all different. The following methods were employed: exergaming, flexibility exercises, physiotherapy, Tai Chi, land and water workouts, and posture re-education (Supplementary Table 1). When it comes to pain and BASDAI, a combination of hydrotherapy and exercise may be more helpful than exercise alone (Altan et al., 2006; Gurcay et al., 2008). Although not always the case, research suggests that group-based treatments have a better chance of success than home-based programs (Cagliyan et al., 2007; Karapolat et al., 2008).

Genetics of ankylosing spondylitis

Historical perspective

Midway through the twentieth century, scientists made great strides in our knowledge of AS when they discovered the HLA-B27 antigen, establishing a hereditary connection to the condition. This ground-breaking finding improved our understanding of the inflammatory processes underlying AS and redirected research efforts towards investigating immunological pathways. Nonsteroidal anti-inflammatory drugs (NSAIDs) were the mainstay of early treatment regimens; however, these drugs only alleviated symptoms and did nothing to stop the disease's progression or address the underlying inflammation [4-6]. Complex and multi-factorial, with a strong hereditary component, AS is a hallmark of the disorder. Understanding the pathophysiology of AS and improving diagnostic and therapeutic methods has necessitated research into the genetic variables linked with the disorder.

HLA-B27 and genetic predisposition

When the HLA-B27 antigen is present, there is the greatest genetic link with AS. In contrast to the general population, which only accounts for roughly 5-10% of AS cases, over 90% of AS patients test positive for this antigen. While additional environmental and genetic variables likely contribute to the disease's development, the presence of HLA-B27 is thought to be a major risk factor.

Other genetic factors

Despite HLA-B27's status as the most well-known AS genetic marker, additional loci have been linked to the disorder in more recent research. Multiple disease-related Single Nucleotide Polymorphisms (SNPs) have been found by Genome-Wide Association Studies (GWAS). Important areas of DNA contain:

1. ERAP1: This gene is responsible for encoding an enzyme that plays a crucial role in the processing of antigens for T cell presentation. Variants within the ERAP1 gene are believed to affect immune responses and have been linked to susceptibility to AS .
2. IL-23R: Genetic variations in the interleukin-23 receptor gene have been associated with AS, underscoring the significance of the IL-23/IL-17 signaling pathway in the inflammatory mechanisms of the disease.
3. Other loci: Numerous additional genes associated with immune function, including IL-1, IL-6, and TNF, have been identified as contributing factors to the susceptibility of AS through findings from genome-wide association studies (GWAS).

The T helper 17/23 (Th17/23) axis and its associated genetic variants are implicated in a number of inflammatory bowel diseases (IBD), psoriasis, and AS, according to genome-wide association studies. All of this points to a common pathogenic mechanism, and it also suggests that the microbiome might play a role in how these diseases develop.

Pathophysiology of ankylosing spondylitis

An abnormal immune response, particularly in the innate immune system, characterizes AS, and recent research has illuminated its complicated etiology. Though it can affect other peripheral joints and entheses (the places where ligaments and tendons hook to the bone), this autoimmune disorder mostly affects the spine and sacroiliac joints. Additionally, advancements in imaging technology, particularly MRI, have made it easier to detect sacroiliitis and other inflammatory alterations earlier, allowing for more prompt treatments.

Disregulation of the immune system: Certain genetic variables, like the HLA-B27 gene, are associated with AS and play a crucial role in activating the immune system. Although carrying this gene does not ensure that AS will develop, it does increase the risk significantly. Recent research has shown that those who possess the HLA-B27 gene are at a higher risk of experiencing T-cell activation and an abnormal inflammatory response due to immune system interactions with gut microbiome.

Inflammatory cascade key cytokines: A key component of AS pathogenesis is the presence of inflammatory cytokines, including TNF-α and IL-17. Inflammation in the entheses and joints is driven in large part by these cytokines.

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1. Bone remodeling: erosion and new bone formation

A hallmark of AS is a process known as bony remodeling, which includes both the loss and replacement of bone. In this procedure, immune cells like neutrophils and macrophages are able to penetrate the damaged joints when the innate immune system is activated. The activation of osteoclasts, which leads to bone resorption, is caused by the pro-inflammatory cytokines produced by these cells, such as TNF and IL-17. Simultaneously, the enthesis stimulates osteoblasts to produce new bone, which might induce the development of syndesmophytes, which in extreme cases can lead to spinal fusion and a marked decrease in mobility.

Microbiome and ankylosing spondylitis

New research is beginning to shed light on the link between the gut microbiome and AS, suggesting that alterations to the microbiota in the gut may influence the development and progression of the condition. A number of autoimmune illnesses, including AS, have been linked to dysbiosis, which is defined as an imbalance in microbial populations, and the gut microbiome plays a crucial role in immune system regulation.

Dysbiosis and immune response:

Based on the research conducted, it has been shown that persons with AS often have dysbiosis. This condition is characterized by an imbalance in the microbiome, where helpful bacteria are reduced and pro-inflammatory bacteria are increased. A potential consequence of this imbalance is an increase in intestinal permeability, which can lead to systemic inflammation and the admission of microbial antigens into the circulation. Any disruptions to this complex system, which includes the gut microbiota, might cause the immune system to respond inappropriately, which in turn can cause chronic inflammation and the progression of different illnesses. It is thought that dysbiosis significantly affects immunological responses in AS through many pathways. One of the main ways AS develops is by the activation of Th17 cells, which produce interleukin-17 (IL-17), an important pro-inflammatory cytokine. It has been discovered that some microbial species, particularly those in the Enterobacteriaceae family like Klebsiella pneumoniae, can enhance IL-23 production and thereby promote Th17 differentiation. As a result, Th17 cells are further activated. The inflammatory response in peripheral joints and entheses is intensified by the production of additional pro-inflammatory cytokines, such as Tumor Necrosis Factor (TNF), by these activated Th17 cells. This leads to the pain, stiffness, and joint degeneration that are characteristic symptoms of AS.

Inflammatory cytokines:

An important part of the pathophysiology of AS is the generation of inflammatory cytokines by certain gut bacteria, such as Tumor Necrosis Factor (TNF) and interleukin-17 (IL-17). The disease's activity can be affected by the presence or absence of certain bacterial species, which in turn affects cytokine production. When HLA-B27 interacts with the microbiota, it sets off a cascade of events that causes inflammation throughout the body, beginning in the intestines and spreading to other organs, including the joints. This pathway greatly contributes to the inflammation seen in AS by causing immune cells to release inflammatory cytokines like IL-17 and TNF in response to cues from microbes. Dysbiosis, in which the intestinal barrier is compromised due to an imbalance in gut microbiota, amplifies this inflammatory response because microbial components, such as lipopolysaccharides (LPS), are able to enter the circulation and cause systemic inflammation. High levels of inflammation have been linked to this disorder, which is often known as "leaky gut," in individuals with AS. New treatment strategies to address microbial imbalances and modulate immune responses in AS may be possible when we learn more about the role of HLAB27 and the microbiome in impacting systemic inflammation.

HLA-B27 and the microbiome:

It is known that the HLA-B27 antigen is a major hereditary risk factor for AS. According to new research, the microbiome may activate inflammatory pathways that aid in disease development through interactions with HLA-B27, which in turn affect immunological responses. In an effort to better understand the causes of AS, researchers have focused heavily on the role that the gut microbiota plays in connection to HLA-B27. More than 90% of people with AS have the HLA-B27 allele, making it a major genetic risk factor for the disease. New research suggests that HLA-B27 may influence the gut microbiota's make-up and activity, which might lead to systemic inflammation. Some bacterial families, such Enterobacteriaceae, which includes Klebsiella pneumoniae, tend to proliferate in the gut microbiome of people who carry the HLA-B27 gene. A key mechanism in the pathophysiology of AS is Th17 differentiation, and it is thought that these bacteria promote immune activation by easing this process. In those who are predisposed to AS genetically, this dysbiosis may create an environment in the microbiome that supports immunological responses.

Therapeutic implications:

Discovering how the microbiota contributes to AS opens the door to new treatment possibilities. Traditional therapies may be supplemented with probiotics and dietary changes to restore a normal gut flora, which may reduce inflammation and improve patient outcomes. Animal studies have also shown that HLA-B27 is insufficient for AS to develop on its own; for example, transgenic rats reared in an environment devoid of germs did not develop spondyloarthritis (SpA). The quest for potential infectious agents that cause AS has recently taken center stage in the scientific community. It is believed that Klebsiella pneumoniae, the first bacteria to be mentioned in this context, has an antigen similar to one generated by the HLA-B27 gene. But the exact process is still a mystery, and some research suggests it probably has little to do with AS. The following bacterial families are also linked to AS: Bacteroidaceae, Lachnospiraceae, Prevotellaceae, Rikenellaceae, and Porphyromonadaceae. Patients with periodontal disease had higher levels of antibodies against Porphyromonas gingivalis and Prevotella intermedia, suggesting that periodontal disease and other non-gut bacteria may have a role in the development of SpA. There is substantial spine dysmobility in AS, and some research suggests

a connection between chronic periodontitis and this condition. New research has shown mixed findings in this area, therefore it's still being studied (Table 1).

Table 1: A comprehensive review of present and prospective therapy possibilities for AS, with an emphasis on therapeutic methods that take into account the microbiome, bacterial infections, and immunological pathways.

Treatment Approach	Description	Potential Impact on AS	Supporting Evidence
Probiotics	Supplementation with beneficial bacteria to restore gut microbiota balance.	Mayreducesystemicinflammationandimproveguthealth,potentiallyalleviating AS symptoms.	Studies suggest that probiotics may have anti-inflammatory effects and modulate immune responses in autoimmune diseases .
Dietary Modifications	Changes in diet to favor gut health, such as increased fiber intake or reduced processed foods.	Aimed at enhancing micro biome diversity, reducing inflammation, and potentially improving AS outcomes.	Dietary interventions have been linked to changes in gut microbiota composition, with potential benefits in autoimmune diseases .
Antibiotics	Targeting specific bacteria that may contribute to AS pathogenesis (e.g., Klebsiella pneumoniae).	Could potentially reduce bacterial load associated with AS, particularly in the gut and oral cavity.	Evidence of a link between specific pathogens (e.g., Klebsiella) and AS, though the role of antibiotics is still under investigation .
TNF Inhibitors	Drugs such as infliximab and etanercept that target TNF, a pro-inflammatory cytokine.	Directly reduces systemic inflammation, often leading to significant improvements in AS symptoms.	Well-established as a first-line biologic therapy for AS with strong evidence of efficacy in reducing inflammation and improving function.
IL-17 Inhibitors	Agents such as secukinumab that inhibit IL17, a cytokine involved in AS inflammation.	Reduces inflammation and may be particularly effective for patients with peripheral arthritis.	Effective in treating AS, especially in patients who have not responded to TNF inhibitors.
Periodontal Disease Treatment	Oral hygiene interventions, such as scaling and root planing, to reduce periodontal inflammation.	May reduce systemic inflammation and improve spinal mobility in AS patients with periodontal	Some studies suggest a link between chronic periodontitis and spinal involvement in AS .

		disease	
HLA-B27Targeting	Potential future therapies targeting HLAB27 expression or its interaction with the microbiome.	Could reduce the genetic predisposition to AS by modulating immune responses to microbial antigens.	Animal studies suggest that HLA-B27 alone is insufficient for AS onset; environmental factors (e.g., microbiome) likely play a role.

Gender differences in ankylosing spondylitis

As a chronic inflammatory condition, AS mostly affects the pelvis and spine. There are substantial gender differences in its prevalence, clinical manifestations, and response to therapy, according to the available research.

1. Prevalence: According to the data, AS is more commonly seen in males than in women, with a male-to-female ratio ranging from 2:1 to 4:1. This variation may indicate that the condition's risk is influenced by hormonal or genetic factors. The immune-boosting effects of estrogen have long been thought to contribute to the higher prevalence and severity of autoimmune illnesses like AS in women. There is speculation that estrogen may influence the inflammatory pathways linked to AS by regulating cytokine production and reorienting the immune response towards a Th2 profile. On the flip side, testosterone is thought to have immune-suppressing effects, which might explain why male patients with the condition tend to have a slower progression. This is because testosterone is generally found in higher quantities in males.
2. Clinical presentation: Symptoms experienced by men and women are frequently distinct. Peripheral arthritis is more common in women and spinal problems may be less severe in women. On top of that, they are at a higher risk of experiencing symptoms like lethargy and enthesitis, which might cause them to miss a diagnosis (26). It is unclear why women with AS take longer to get a diagnosis than men do; this might be because of variations in symptoms or prejudice on the part of clinicians. Women also tend to have more severe illness symptoms, according to the research. This includes increased involvement of the peripheral joints and higher levels of functional impairment.
3. Disease progression: Spinal problems are more common in males, according to the available evidence, while women may exhibit more severe functional impairment. In addition, comorbidities like anxiety and depression might manifest differently in men and women, which can impact health outcomes generally.
4. Treatment response: Treatment outcomes can differ across sexes, especially when it comes to biologic medicines. There is some evidence that women may have a more favorable reaction to some drugs, such TNF inhibitors, but they may also experience more severe side effects than males. According to research, TNF inhibitors may have a more favorable effect on women, but IL-17 inhibitors seem to have a more consistent effect on both sexes. The clinical significance of gender discrepancies in treatment results has been underscored by integrating this information.

The role of environmental factors

Environmental variables have a substantial influence on the start and course of AS, in addition to genetic factors, which are critical. Potential causes include infections, mechanical stress, and the makeup of the gut flora. To further understand why some people with the HLA-B27 antigen do not develop AS, researchers are focusing on the possible role of environmental factors in connection to genetic predispositions.

Therapies for ankylosing spondylitis

Inflammatory arthritis, of which AS is a subtype, mainly manifests in the spine. Symptom relief, mobility enhancement, and complication prevention are the usual goals of treatment (Tables 2 and 3). These are a few typical treatments:

Medications:

Nonsteroidal anti-inflammatory drugs (NSAIDs): Ibuprofen and naproxen are two examples of over-the-counter pain relievers and anti-inflammatory drugs.

Disease-modifying antirheumatic drugs (DMARDs): When it comes to peripheral arthritis, agents such as sulfasalazine really shine.

Biologics: For more advanced instances, treatments like IL-17 inhibitors (e.g., secukinumab) and TNF inhibitors (e.g., adalimumab, etanercept) work.

Corticosteroids: These can be used to manage temporary flare-ups.

Physical therapy:

Maintaining mobility and good posture requires consistent physical activity and treatment. Strength training for the back and other specific muscles can improve mobility and performance.

Exercise:

A fundamental component of any effective workout program should include a regular schedule of stretching and strengthening exercises. Among the many health benefits of exercise, swimming and yoga stand out (33).

Lifestyle modifications:

Improving one's posture, quitting smoking, and reaching and maintaining a healthy weight are all great ways to alleviate symptoms.

Heat and cold therapy:

Muscle stiffness and relaxation can be achieved with heat treatment, while inflammation can be efficiently reduced with cold therapy.

Alternative therapies:

Acupuncture, massage, and other alternative therapies may help some people, but the research on their efficacy is mixed.

Surgery:

Surgical procedures, such as joint replacement, may be considered in extreme circumstances involving damaged joints. The best way to monitor the disease's course and make adjustments to treatment programs is to have regular appointments with a rheumatologist.

Table 2: Therapies for Ankylosing spondylitis.

Therapy Type	Description	Examples/Details
Medications	Focuses on reducing inflammation and modulating the immune response.	NSAIDs (e.g., ibuprofen, naproxen), DMARDs (e.g., sulfasalazine), Biologics (TNFinhibitors:adalimumab,etanercept;IL-17inhibitors:secukinumab),Corticosteroids.
Physical Therapy	Improves flexibility, posture, and mobility	Targetedexercisestostrengthenbackmusclesandmaintain functionality.
Exercise	Enhances physical health and reduces stiffness.	Swimming, yoga, and stretching exercises.
Life style Modifications	Addresses overall health factors.	Healthy weight, stopping smoking, good posture.
Heat and Cold Therapy	Provides relief from stiffness and inflammation.	Heat for muscle relaxation, cold for inflammation reduction.
Alternative Therapies	Complementary methods for symptom management.	Acupuncture, massage, and other alternative treatments.
Surgery	Considered in severe cases where other treatments are ineffective.	Joint replacement or surgical interventions for damaged joints.

Patient-centered approaches

A holistic strategy for patient care is becoming more important in view of recent developments. To treat the physical and mental impacts of AS, it is vital to have multidisciplinary teams that include rheumatologists, physiotherapists, and psychologists. Moreover, self-management techniques and

patient education are being recognized as essential components of treatment that empower individuals to actively engage in their own health care.

Conclusion

Spinal mobility, physical function, and quality of life are all profoundly impacted by Ankylosing Spondylitis, a chronic inflammatory illness. Beyond pharmaceutical treatment, a thorough and interdisciplinary strategy is necessary for the effective care of this condition. Modifying one's lifestyle has grown in importance as an integral part of managing chronic diseases, alongside medicine, which is essential for lowering inflammation and symptoms. This clinical study's results show that patients can benefit greatly from making planned modifications to their lifestyle. Spinal flexibility, discomfort, and stiffness were all improved with regular exercise, physiotherapy, and posture correction, leading to greater functional capability. Prevention of spinal abnormalities and maintenance of mobility can be achieved by low-impact activities including swimming, breathing exercises, and stretching. Improved respiratory function and general physical endurance are additional benefits of these workouts. In addition to helping with overall health, eating a healthy, well-rounded diet may also help with inflammation management. Patients with ankylosing spondylitis can improve their physical and mental health by adopting good lifestyle choices including not smoking, keeping a healthy weight, getting enough sleep, and managing stress effectively. In addition to alleviating symptoms, these interventions improve patients' capacity to carry out everyday tasks and keep their independence. In conclusion, the study highlights the fact that patients with ankylosing spondylitis can greatly benefit from better disease management and quality of life when conventional medical therapy is combined with lifestyle modification tactics. As part of standard treatment, medical providers should push their patients to make good eating, regular exercise, and proper posture a priority. Further validation of lifestyle intervention efficacy and development of standardized therapeutic recommendations for holistic care of ankylosing spondylitis should be pursued through future research utilizing bigger populations and long-term follow-up.

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