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RESEARCH ARTICLE

Effect of Integrated Yoga Therapy on Physical Function in Patients with Osteoarthritis

Dr. Bijendra Singh[1*], Dr. Lata Sati[2], Dr. Kanchan Joshi[1], Dr. Anil Thapliyal[1], Dr. Surendra Parsad Rayal[1], Dr. Saraswati Kala[1] and Dr Savita P Patil[1]

[1]School of Yogic sciences & Naturopathy, Shri Guru Ram Rai University, Dehradun Uttarakhand (India),
[2]Department of Home Science, School of Humanities and Social Sciences, Shri Guru Ram Rai University, Dehradun, Uttarakhand (India).

*Corresponding Author
Dr. Bijendra Singh
(gusainbijendra@gmail.com)

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Abstract: The most prevalent kind of arthritis, osteoarthritis (OA), is characterised by the gradual deterioration of joint cartilage, which causes pain, stiffness, and restrictions in one's ability to operate. Physical therapy and pharmaceutical treatments are examples of conventional care; nevertheless, these methods sometimes provide only little alleviation and may have adverse consequences. Complementary therapies that increase physical function, lessen pain, and improve general quality of life include integrative techniques like yoga therapy. ***Objective***: The purpose of this study was to assess how integrated yoga treatment affected the physical function, level of pain, and overall quality of life of patients with osteoarthritis in their knees. ***Mythology***: Based on the Yoga Centre criteria of Dehradun (India), 40 volunteers with knee osteoarthritis, ages 40 to 60, participated in a randomised controlled experiment. The participants were split into two groups at random: the control group (n=20) got regular physiotherapy, while the intervention group (n=20) received integrated yoga treatment. For ninety days and forty-five minutes, the yoga intervention comprised a mix of asanas (postures), pranayama (breathing methods), and guided relaxation. The visual analogue scale (VAS) was used to quantify the severity of pain, and the SF-36 questionnaire was used to examine quality of life. Assessments were carried out both before and after the intervention. ***Result***: Analysis conducted after the intervention revealed that the yoga group had significantly improved when compared to the control group. A higher decrease in WOMAC scores ($p < 0.01$) was seen in the yoga group, suggesting better physical function.

Keyword: Osteoarthritis, Yoga therapy, Integrated approach, Pranayama.

INTRODUCTION

The most common kind of arthritis, osteoarthritis (OA), affects millions of individuals globally, especially the elderly. Degradation of articular cartilage, alterations in subchondral bone, inflammation of the synovium, and decreased joint function are the hallmarks of this chronic, progressive joint disease. Weight-bearing joints such the hands, knees, hips, and spine are the main targets of OA. It is essential to comprehend the physiological and anatomical alterations associated with OA in order to diagnose, treat, and stop future decline.

The following essential structures make up a synovial joint, which is the one most frequently impacted by OA:

In synovial joints, the smooth, white tissue covering the ends of bones is called articular cartilage. It serves as a cushion and lessens friction when moving. Cartilage is composed mostly of water (70–80%), collagen (primarily type II), and proteoglycans (aggrecan). It is a neural (no nerves) and avascular (no blood flow).

The layer of bone directly beneath the cartilage is called the subchondral bone. It absorbs shock and supports the cartilage on top. A viscous substance that lubricates and nourishes avascular cartilage is called synovial fluid. Ligaments and joint capsules: they offer stability and defence for joints. Ligaments link bones and direct joint mobility, while the capsule encloses the joint cavity. Tendons connect muscles to bone, which helps to stabilise joints, while surrounding muscles regulate mobility.

The pathogenesis of osteoarthritis: More than just "wear and tear," osteoarthritis is the consequence of a complex interaction between inflammatory, metabolic, and mechanical processes. Osteophyte production, subchondral bone remodelling, increasing cartilage degradation, and synovial inflammation are all part of the pathogenesis. In the early phases of osteoarthritis development, relatively modest biochemical changes take place, despite the fact that osteoarthritis is a degenerative joint disease that can result in gross cartilage loss and morphological damage to other joint components. Healthy cartilage has a precisely balanced water content, with hydrostatic and osmotic pressures drawing water in and compressive force pushing water out. While cartilage proteoglycans and the Gibbs-Donnan effect provide osmotic pressure, which tends to suck water in, collagen fibres provide the compressive force. However, the collagen matrix gets more jumbled and the amount of proteoglycan in cartilage decreases as osteoarthritis progresses. Water content rises as a result of the disintegration of collagen fibres. This rise happens because the loss of collagen outweighs the total loss of proteoglycans, which results in a lower osmotic pull.

It may also have an impact on other joint structures. The menisci may deteriorate and wear away, and the ligaments inside the joint may thicken and fibroticize. By the time a person has a joint replacement, menisci may be totally gone. In the absence of the menisci, new bone outgrowths known as "spurs" or osteophytes may develop on the joint edges, maybe in an effort to increase the congruence of the articular cartilage surfaces. The amount of subchondral bone grows as the mineralisation decreases (hypo mineralisation). All of these modifications may result in functional issues. Thickened synovium and subchondral bone lesions have been linked to osteoarthritic joint discomfort.

Unlike T-cell activation in the joint lining of individuals with rheumatoid arthritis, osteoarthritis is characterised by the involvement of macrophages through the activation of the innate immune system, which results in inflammation of the joint lining (synovium).In osteoarthritis, pro-inflammatory cytokines activate matrix metalloproteinases, causing joint remodelling and destruction. The process is fuelled by the production of inflammatory cytokines in response to tissue injury or deterioration of the synovium or articular cartilage.

Figer 1: pathophysiology Osteoarthritis

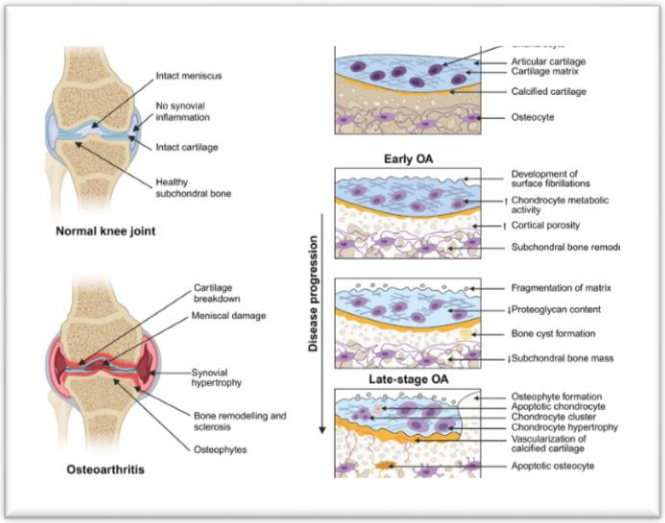


Table 1: The Integrated Model of the Pathophysiology of OA

Domain	Key Mechanisms	Examples / Details
Mechanical Elements	Obesity, injury, malalignment, repeated stress, and abnormal joint loading	Meniscal tears, valus/valgus abnormalities, and elevated BMI that result in higher knee load
Pathology of Cartilage	ECM deterioration, proteoglycan loss, collagen disintegration, and chondrocyte death	MMPs (MMP-1, MMP-13) and ADAMTS (aggrecanase) activities are upregulated.
Bone Changes	Subchondral bone sclerosis, bone marrow lesions, osteophyte formation	Increased bone turnover, altered biomechanics due to osteophytes
Inflammation of the Synovium	Release of cytokines, invasion of immune cells, and synovitis	IL-1β, IL-6, and TNF-α increase pain sensitivity and catabolic activity
Ligaments and the Meniscus	Laxity, tears, and degeneration all contribute to instability and changed mechanics.	Damage to the cruciate ligament and meniscal extrusion
Molecular Mediators	Growth factors, oxidative stress, chemokines, and pro-inflammatory cytokines	VEGF, prostaglandins, ROS, and NF-κB activation

Mechanisms of the Nervous System	Changes in pain processing and peripheral and central sensitisation	Chronic pain development and synovial/subchondral bone nociceptor activation
Metabolic and Systemic Factors	Adipokines, ageing, metabolic syndrome, and low-grade inflammation	Advanced glycation end-products (AGEs); dysregulation of leptin, resistin, and adiponectin
Genetic and Epigenetic Variables	Epigenetic modulation of cartilage homeostasis and genetic vulnerability	COL2A1 and GDF5 polymorphisms; changed DNA methylation and microRNAs
Integrated Result	Systemic variables, molecular mediators, and biomechanical stress interact to cause progressive OA alterations.	Joint deformity, pain, stiffness, and a loss in function

physical function in patients with osteoarthritis: Millions of people worldwide suffer from osteoarthritis (OA), the most prevalent chronic joint disease, especially in older age. Osteophyte production, subchondral bone remodelling, gradual cartilage loss, and variable levels of synovial inflammation are its defining features. Although the illness is primarily caused by structural deterioration, OA's effects on pain, stiffness, and physical function are its most incapacitating effects. Because it has a direct impact on a person's independence, capacity to carry out daily tasks, and quality of life, physical function is a critical outcome in OA. Effective care of OA patients requires an understanding of the changes in physical function, the causes driving it, and strategies to increase function.

Physical Function's Significance in OA: The capacity to carry out actions and activities that facilitate everyday life, movement, and social interaction is referred to as physical function. Pain, joint deformity, muscular weakness, stiffness, and exhaustion are among symptoms of OA that affect physical function. Patients frequently have difficulties with both instrumental activities of daily living (IADL) like cooking, shopping, or transportation, as well as fundamental ADLs like walking, dressing, or bathing. Dependency, social isolation, and psychological suffering can result from OA patients' declining physical function.

Limitations in Physical Function in OA:
The joint in question determines the precise functional limitations:
Knee Osteoarthritis: difficulty climbing stairs or walking great distances. stiffness and pain when getting out of a chair or toilet. diminished capacity for cross-legged sitting, kneeling, and squatting.

- Pain and weakness in the quadriceps cause alterations in gait, which frequently lead to instability.
- Hip Osteoarthritis: difficulties walking, particularly on rough terrain.
- restricted hip flexion and rotation, which makes it difficult to put on socks or shoes.
- difficulty getting in and out of automobiles or sitting on low seats.
- limping as a result of discomfort and constricted joints.
- Osteoarthritis of the Hand and Fingers: diminished fine motor abilities and grip strength.
- difficulty doing dexterity-demanding chores like writing, buttoning clothing, or opening jars.
- Precision tasks are hampered by deformities such as Heberden's and Bouchard's nodes.
- Osteoarthritis of the spine: Pain and stiffness make it difficult to bend, twist, and lift. issues with extended sitting, walking, or standing.
- disruption of sleep brought on by back discomfort.
- Physical Function-Related Factors: Structural deterioration alone does not dictate physical function in OA. It is affected by several interrelated factors:
- Severity of pain: The best indicator of impairment is pain. It causes muscular weakness, decreases mobility, and heightens anxiety of exertion.
- Joint stiffness: Slow movement is caused by stiff joints in the morning or after inactivity.

- Muscle weakness: In knee OA, quadriceps weakness increases impairment, while in hand OA, grip weakness increases disability.
- Joint malalignment: Advanced hip abnormalities or varus or valgus deformities in the knee affect mobility and biomechanics.
- Obesity: Carrying too much weight puts more strain on joints, making discomfort worse and reducing range of motion.
- Psychological factors: Fear-avoidance attitudes, anxiety, and depression can aggravate disability and decrease physical activity.
- Comorbidities: Limitations are exacerbated by neurological issues, diabetes, or cardiovascular illness.

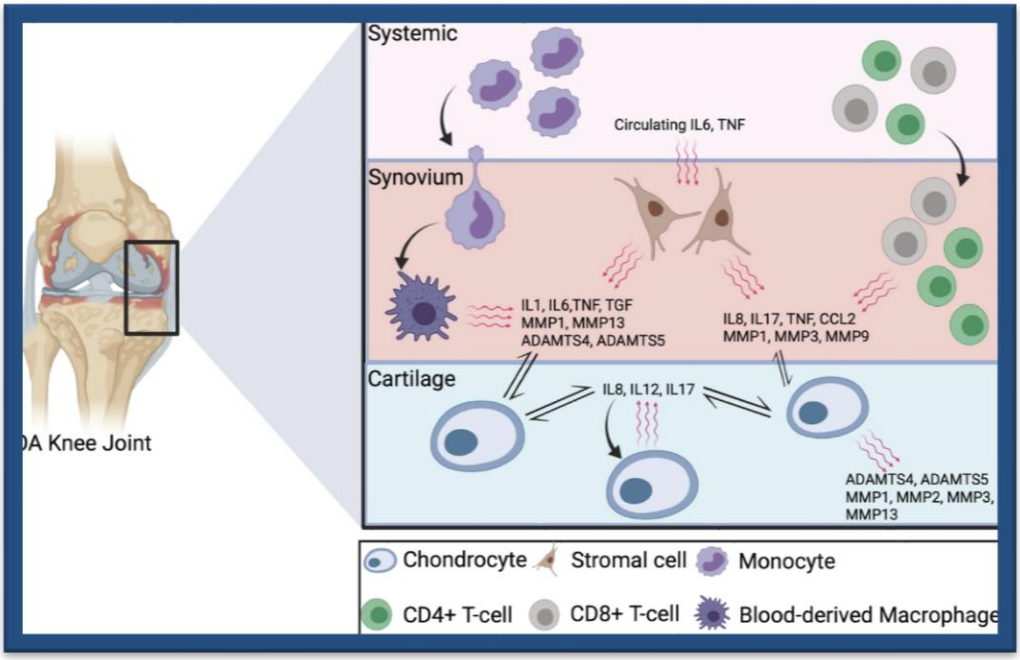
Mechanisms of Osteoarthritis: The most common chronic joint disease is osteoarthritis (OA), which is typified by the gradual loss of articular cartilage, alterations in subchondral bone, the production of osteophytes, inflammation of the synovium, and a reduction in function. OA is now understood to be a complex illness involving mechanical, biochemical, inflammatory, metabolic, and hereditary causes, rather than a straightforward "wear and tear" syndrome. Developing therapeutic therapies, rehabilitation programs, and preventative efforts all depend on an understanding of these systems. Articular cartilage in a healthy joint offers a low-friction, smooth surface that makes movement pleasant. In order to preserve its structural integrity and resilience, cartilage is made up of chondrocytes embedded in an extracellular matrix (ECM) that is abundant in type II collagen and proteoglycans. The joint is nourished and lubricated by synovial fluid, while cartilage is supported by the subchondral bone. The equilibrium between chondrocytes' anabolic (repair) and catabolic (degradative) activities, which are controlled by growth hormones, cytokines, and mechanical loads, is essential for maintaining joint homeostasis. This delicate equilibrium is upset in OA, which causes gradual deterioration to the structure and function of the joints.

Unusual Mechanical Stress: Micro-injury in articular cartilage is caused by excessive joint loading brought on by trauma, obesity, malalignment, or recurrent stressing. Matrix disruption, chondrocyte death, and the release of matrix fragments into the synovial space are the outcomes of this.

Degradation of Cartilage: Due to its lack of a direct blood supply, cartilage is not very regenerating. Abnormal loading reduces cartilage's resistance to compression by rupturing collagen fibrils and causing the loss of proteoglycans. Eventually, the surface erodes and becomes soft and fibrillated.

Changes in Subchondral Bones: More stress is transmitted to the subchondral bone as a result of cartilage thinning. This results in the development of cysts, microfractures, and bone sclerosis. Osteophyte production at joint borders as a result of adaptive remodelling may stabilise the joint but may also cause deformation.

Figer 2: Mechanisms of Osteoarthritis



Molecular and Cellular Processes: Chondrocyte function changes from a homeostatic to a catabolic state at the cellular level in OA.

Dysfunction of Chondrocytes: Chondrocytes typically maintain a balance between ECM production and breakdown. They alter phenotypically and release more degradative enzymes when they have OA. Reduced reparative ability is a result of chondrocyte apoptosis.

Enzymes that Degrade Matrix: Type II collagen is broken down by matrix metalloproteinases (MMPs), specifically MMP-1, MMP-3, and MMP-13. Aggrecan, a significant proteoglycan, is broken down by ADAMTS, a disintegrin and metalloproteinase with thrombospondin motifs, particularly ADAMTS-4 and ADAMTS-5. Cartilage degeneration is accelerated by an imbalance between matrix production and degradation.

Cytokines that promote inflammation: The three main mediators that chondrocytes and synovial cells generate are IL-1β, TNF-α, and IL-6. They promote the release of prostaglandins and nitric oxide, block the synthesis of type II collagen, and stimulate the formation of MMP and ADAMTS. This leads to a vicious cycle of cartilage deterioration and inflammation.

Stress Due to Oxidation: DNA and cartilage proteins are damaged by reactive oxygen species (ROS). When chondrocytes have mitochondrial malfunction, their anabolic capacity and viability are diminished.

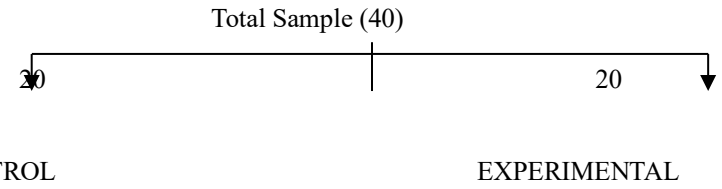
Inflammation of the Synovium (Synovitis): Despite being predominantly a degenerative disease, inflammation is a major factor in OA. Damage-associated molecular patterns (DAMPs) and cartilage fragments in the synovial fluid cause synovitis. Growth factors, chemokines, and cytokines produced by synovial cells prolong joint injury. Chronic, low-grade inflammation exacerbates functional impairment by causing discomfort, stiffness, and oedema.

Integrated Approach to Osteoarthritis Management: The gradual deterioration of articular cartilage, synovial inflammation, subchondral bone remodelling, and functional impairment of afflicted joints are the hallmarks of osteoarthritis (OA), a complex, multifactorial disease. OA, once thought to be a purely mechanical "wear-and-tear" syndrome, is now understood to be a disorder impacted by a combination of genetic, physiological, metabolic, mechanical, and psychological variables. Because of this complexity, single-modality therapies frequently fall short in meeting the multifaceted demands of individuals with OA. Therefore, a more thorough, patient-centred approach to addressing OA is provided by an integrated approach that incorporates biological therapies with physiotherapy, yoga, diet, lifestyle adjustment, and psychological support. Pharmacological therapies include corticosteroid injections, nonsteroidal anti-inflammatory medications (NSAIDs), and, in extreme situations, joint replacement surgery are frequently the mainstays of conventional OA therapy. Although these methods alleviate symptoms, they seldom ever address the lifestyle and systemic factors that contribute to the evolution of the disease. Furthermore, long-term drug usage is linked to negative side effects, such as cardiovascular, renal, and gastrointestinal concerns. Conversely, complementary approaches like yoga, physiotherapy, and lifestyle changes focus on inflammation, joint function, muscular strength, and coping methods for patients, offering comprehensive advantages with negligible adverse effects. Therefore, combining biological treatments with lifestyle, psychological, and rehabilitative modalities guarantees that the therapy not only improves quality of life and slows the course of the disease, but also relieves symptoms. The foundation of OA treatment is biomedical management, especially in the early stages when the main objective is symptom reduction. To lessen pain and inflammation, pharmacological therapy includes the prescription of NSAIDs, paracetamol, and selective COX-2 inhibitors. Intra-articular corticosteroid injections are utilised for individuals who experience significant flare-ups, and hyaluronic acid injections are taken into consideration for Visco supplementation. In more severe situations when conservative therapies are ineffective, surgical procedures like arthroscopy, osteotomy, or complete joint replacement are saved until last. Although they are still being researched, biological treatments such as stem-cell and platelet-rich plasma (PRP) are becoming more popular. Despite their effectiveness, biological methods need to be combined with lifestyle and rehabilitation techniques to improve long-term results and avoid reliance.

OA is a psychological issue in addition to a medical illness. Depression, anxiety, social disengagement, and a worse quality of life are frequently caused by chronic pain. Therefore, behavioural and psychological therapies are essential components of an integrated care approach. Patients who get cognitive-behavioral therapy (CBT) are able to change their unfavourable perceptions of pain and impairment. Patient education initiatives provide people with self-management skills, including pacing exercises, joint-protection procedures, and at-home workouts. Counselling and support groups improve adherence to treatment plans, lessen feelings of loneliness, and offer emotional comfort. This domain guarantees that OA sufferers cultivate resilience and a positive mindset in addition to managing their physical symptoms.

Patient and Methodology

The sample of 40 patients of Effect of integrated yoga therapy on physical function in patients with osteoarthritisThey have been further dived into control group & experimental group. Sample will be selected from Dehradun City (Uttarakhand) belonging to age group between 40 to 60 years. The subjects were selected by using quota sampling technique.



Yogic Intervention for the Present Study- For a predetermined amount of time, the experimental group participated in a structured comprehensive yoga treatment program, whereas the control group received no yoga intervention.

Time Duration for Yogic Practice- 60 Day (50) Min
For a predetermined amount of time, the experimental group participated in a structured comprehensive yoga treatment program, whereas the control group received no yoga intervention.

Table 2: Yogic Intervention

Activity	Time Duration
Pawanmuktasana series 1-Light joint motions for the limbs, both upper and lower	20min
Yogasanas- Asanas to increase mobility and flexibility Tadasana Kati chakrasana Setu Bandhasana Paschimottanasana Marjariasana Ardha Matsyendrasana bhujangasana	10 min
Pranayama-Breathing techniques to promote circulation and relaxation Anulom Vilom Bhramri Bhastrika	10 min
Meditation -Mindfulness and relaxation training	10 min

RESULT:

Figer 3: Paired Samples Statistics

Paired Samples Statistics

	Mean	N	Std. Deviation	Std. Error Mean
Control	87.6000	20	1.53554	.34336
Experimental	39.5000	20	1.46898	.32847

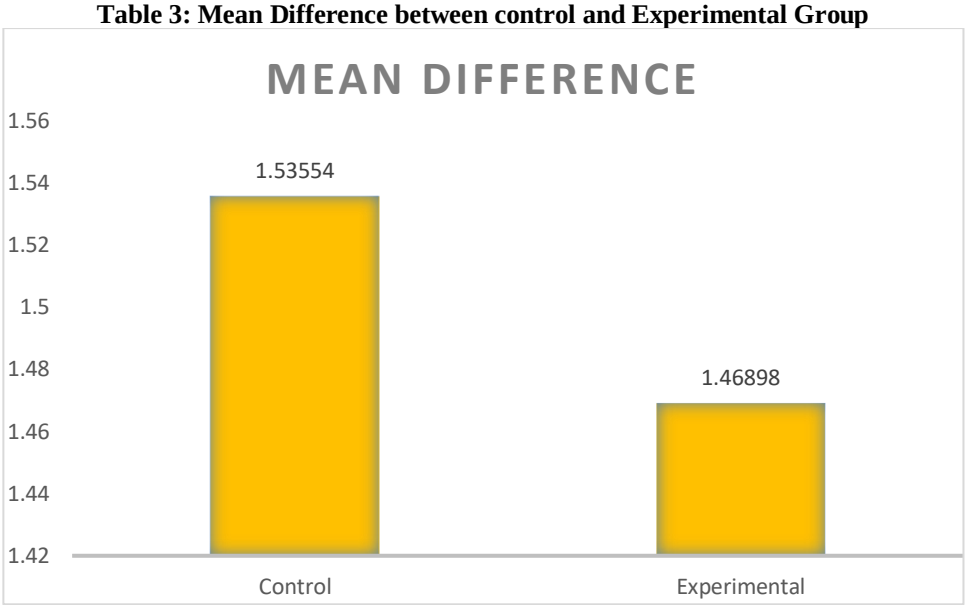
Paired Samples Correlations

	N	Correlation	Sig.
Control & Experimental	20	.350	.130

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
Control & Experimental	48.10000	1.71372	.38320	47.29795	48.90205	125.522	19	.001

Table 3: Mean Difference between control and Experimental Group



The purpose of this study was to assess how integrated yoga treatment affected the physical function of OA patients. Below are the findings from the examination of physical function scores in the experimental and control groups. With a mean score of 87.60, a standard deviation (SD) of 1.53, and a standard error of the mean (SEM) of 0.34, the control group (n = 20) performed well. With an SD of 1.46 and SEM of 0.32, the experimental group (n = 20) that got the integrated yoga therapy intervention, on the other hand, displayed a significantly lower mean score of 39.50. When comparing patients in the yoga treatment group to those in the control group, the large difference in mean values between the two groups indicates a considerable increase in their functional capacity.

The experimental group's lower physical function score suggests improved mobility, less stiffness, and a greater ability to carry out everyday tasks. The significant drop in the experimental group illustrates the beneficial therapeutic effect of the integrated yoga module, since higher scores indicate more functional impairment. However, the control group's score remained quite high, indicating that osteoarthritis-related functional impairments continued in the absence of yoga therapy. The small standard deviations (1.53 in the control group and 1.46 in the experimental group) suggested that variability was quite low in both groups, according to further research. This uniformity between individuals shows that the observed differences are unlikely to be the result of chance and enhances the findings' dependability.

DISCUSSION

Although not covered in length here, a statistical comparison (t-test or equivalent) between the two groups would probably show that there is a very significant difference in the mean physical function scores. This supports the claim that integrated yoga therapy is a statistically sound and clinically useful adjunct intervention for the treatment of osteoarthritis. These, the findings unequivocally lend credence to the idea that yoga can significantly lower disability and enhance quality of life for osteoarthritis patients. These results are consistent with past research showing yoga to be a successful non-pharmacological treatment for long-term musculoskeletal disorders.

Degeneration of cartilage, inflammation, discomfort, and reduced functional ability are the hallmarks of osteoarthritis, a degenerative joint disease. Pharmacological treatments such analgesics, non-

steroidal anti-inflammatory medications (NSAIDs), and in extreme situations, surgery, are frequently used in conventional care. Although these therapies could alleviate symptoms, they are frequently linked to adverse consequences, financial strain, or insufficient functional recovery. As a result, it is widely acknowledged that safe and efficient complementary treatments are necessary. Yoga's comprehensive approach to treating the physical and psychological elements of chronic illness has made it a viable intervention in this setting.

There are other ways to account for the experimental group's improvement. Asanas can help reduce mechanical stress on osteoarthritic joints by improving alignment, strengthening supporting muscles, and increasing joint mobility. The circulation of synovial fluid, which aids in joint lubrication and cartilage feeding, is facilitated by mild stretching exercises. Additionally, yoga enhances balance and proprioception, two abilities that are frequently impaired in people with osteoarthritis.

It's possible that pranayama practices like Anulom Vilom and Bhramari helped people cope with stress and increase their pain threshold. It is well known that stress makes osteoarthritis patients feel more disabled and in pain. Yoga lowers sympathetic overactivity and increases parasympathetic activation by regulating the autonomic nervous system, which lessens pain sensitivity and improves general wellbeing. In addition to promoting psychological resilience, meditation and relaxation techniques assist patients in managing their functional limitations and chronic pain.

The results of this investigation align with previous studies. Yoga therapies have been shown in several clinical studies and systematic reviews to enhance physical function, pain, and quality of life in people suffering from musculoskeletal disorders, such as rheumatoid arthritis and osteoarthritis. For instance, research on osteoarthritis in the knee has shown that yoga significantly reduces stiffness, walking time, and functional independence. This bolsters the claim that, in addition to traditional therapy, yoga-based interventions may be incorporated into rehabilitation programs.

The study's low degree of variability, as seen by the narrow standard deviations, is another significant feature. This supports the validity and generalisability of the results by showing that the beneficial effects of yoga were constant among individuals. It should be mentioned, nonetheless, that the research sample size was somewhat small (n=40), and the length of the intervention would make it more difficult to evaluate long-term benefits. The evidence foundation would be strengthened by future studies with bigger sample numbers, longer follow-up times, and multicentre trials.

CONCLUSION

The current study shown that integrated yoga treatment significantly improved physical function in individuals with osteoarthritis compared to the control group. The experimental group exhibited significantly decreased functional impairment scores, which were correlated with improved joint mobility, reduced stiffness, and an enhanced capacity to perform daily tasks. These findings provide strong evidence that, when used thoroughly and methodically, yoga may be an effective non-pharmacological therapy for osteoarthritis.
In contrast to traditional treatments, which mostly concentrate on symptom relief, yoga takes into account mental, emotional, and physical aspects of wellness. By combining asanas, pranayama, meditation, and relaxation techniques, participants were able to increase their general well-being as well as their functional capacity. Yoga's therapeutic promise in the management of chronic musculoskeletal problems is further supported by the consistency of results among individuals.

The results demonstrate the viability and advantages of integrating yoga into regular osteoarthritis care, despite the study's limitations, which included a small sample size and a brief intervention period. To validate these findings and investigate the processes behind yoga's influence on joint health, more extensive and long-term research is necessary. To sum up, integrated yoga treatment is a safe, economical, and comprehensive way to help people with osteoarthritis improve their physical function. In this expanding patient group, its integration into clinical rehabilitation programs and community health efforts may improve quality of life and lessen the burden of impairment.

Contributions from the authors AS: formal analysis, data curation, research, validation, and original draft writing. Project management, supervision. The manuscript was conceptualised, reviewed, and edited with input from all writers. The final version has been read and approved by all of them.

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