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Original Research Article

# Therapeutic Potential of Turmeric in Osteoarthritis: Anti - Inflammatory and Analgesic Perspectives

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**Abstract**

**Background:** Osteoarthritis is associated with inflammation and pain and there is no treatment for its fundamental causes, or known therapies to slow down its progression. Nonsteroidal anti-inflammatory drugs, COX-2 inhibitors, opioid analgesics, corticosteroid injections, hyaluronic acid injections are choice of treatment depending on the severity of disease and patient acceptability. Turmeric and its derivatives have anti-inflammatory activities. Hence the aim of this study is to study the beneficial effects of turmeric in Osteoarthritis.

**Materials and Methods:** This study was a Nested Case Control Study; here Osteoarthritis patients were randomised in two groups using the single blinding method. Each group comprised of 75 patients each, who gave consent for the study. Group I- received turmeric filled capsules - 1 gm tds(total 3 gm /day},Group 2-placebo (protien powder 500mg x tds) for 4 months. This study was undertaken in Dept. of Medicine, Orthopaedics, Era's Lucknow Medical College & Hospital.

**Results:** There was no significant pre-treatment difference between WOMAC scores of groups which were given turmeric and placebo. In intergroup comparison of pain & physical activity (WOMAC Score) between treatment (Group-I) and placebo (Group-II) Group at last visit after 4 months, it was found that the difference in mean WOMAC score between Group I and Group II was found to be highly significant ($p<0.001$). The mean WOMAC score change in Group I was found to be significantly more than the change in Group II. The mean hsCRP level change in Group I was found to be significantly more than the Group II. The mean IL-6 level change in Group I was found to be significantly more than the Group II.

**Conclusion:** Significant reduction in inflammatory markers in osteoarthritis patients after turmeric administration suggests its anti-inflammatory property may have a significant role in symptom relief in osteoarthritis patients. Improvement in WOMAC scores highlights the role of turmeric in symptom alleviation in Osteoarthritis.

**Keywords:** Osteoarthritis, Osteoarthritis Treatment, Therapeutic Potential, Turmeric, Anti-Inflammatory, Analgesic Perspectives, Knee Pain.

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## Introduction

Osteoarhtritis (OA) is a joint disease having multifactorial aetiology. Both systemic factors (e.g. age, sex, genes) and local factors (e.g. muscle weakness, joint deformity) appear to influence the risk of individual joints developing the disease. OA is associated with inflammation and pain and there is no treatment for its fundamental causes, or known therapies to slow down its progression. The major goal of OA treatment is to reduce joint pain induced by inflammation in the joints, daily wear and tear of joints, and muscle strains [1] which can help relieve symptoms, improve ability to move, and allow the patients to stay active. Nonpharmacologic (non-drug) therapies are a key part of OA treatment and are recommended for everyone with OA. These include weight loss, physical therapy, exercise programme, Orthoses, and use of assistive devices [2]. Nonsteroidal anti-inflammatory drugs, COX-2 inhibitors, opioid analgesics, corticosteroid injections, hyaluronic acid injections are choice of treatment depending on the severity of disease and patient acceptability. All the treatment choices have limited efficacy and none of them is free of side effects for long term

use. Joint replacement is a surgical procedure which is expensive and has its own limitations. Dietary supplements, including herbal products, have also been examined for treatment of OA. Several dietary supplements (*e.g.*, glucosamine, glucosamine with chondroitin, devil's claw, S-adenosyl-l-methionine) have demonstrated efficacy compared to placebo [3]. One additional product that has been evaluated and used for treatment of OA is curcuma [4,5]. It has mainly been used as seasonings in many ethnic cuisines in Bangladesh, India, and Pakistan and has long been used as anti-inflammatory treatment in traditional Chinese and Ayurvedic medicines [6]. Turmeric and its derivatives have anti-inflammatory activities. Unlike ginger, turmeric and curcumin do not modulate COX-1 activity [7,8], but modify NF-$\kappa$B signalling, proinflammatory cytokines such as interleukin production and phospholipase A2, COX-2, and 5-LOX activities. Curcumin also modulates the expressions of various transcription factors involved in energy metabolism such as signal transducer and activator of transcription, peroxisome proliferator-activated receptor-$\gamma$, activator protein-1, cAMP responding element binding protein, estrogen response element, and others [8].

As a result, turmeric and its components have been reported to exert beneficial effects on OA, type 2 diabetes, and dyslipidemia. Therefore, the present study was conducted to study the beneficial effects of turmeric in OA.

## Materials and Methods

**Design:** This was a Nested Case Control Study (follow up of 4 months) which was undertaken in Department of Medicine, Orthopaedics, Era's Lucknow Medical College & Hospital. Ethical clearance was taken from the college as well as written informed consent from the patients was taken.

**Participants:** Study subjects were randomised in two groups using the single blinding method. Each group comprised of 75 patients each, who gave consent for the study. Group I- received turmeric filled capsules - 1 gm x tds (total 3 gm /day}, Group 2- placebo (protein powder 500mg x tds) for 4 months. Inclusion criteria comprised, all patients (>35years) having Osteoarthritis, patients who gave consent for the study. Whereas exclusion criteria had patients with joint diseases other than osteoarthritis, pregnant females, acute infective disorders, proven malignancy, critically ill patients, patients with end organ dysfunction.

**Investigations:** WOMAC score at first visit and after 4 months was evaluated for physical activity and pain. Inflammatory markers (IL6, hsCRP) related to osteoarthritis were studied at first visit and after four months in both the groups. Following investigations were done on both group I and group II patients:

- CBC
- ESR
- X ray of the Affected Joint (If patient willing)
- Serum urea
- Serum creatinine
- S. Uric Acid
- FBS (Fasting Blood Sugar)
- Serum Lipid Profile (TG, HDL)
- hsCRP, IL6(to be done twice)

**WOMAC Score**

**The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC)**

| | |
|---|---|
| **Pain** | |
| 1) Walking | 0 1 2 3 4 |
| 2) Stair Climbing | 0 1 2 3 4 |
| 3) Nocturnal | 0 1 2 3 4 |
| 4) Rest | 0 1 2 3 4 |
| 5) Weight bearing | 0 1 2 3 4 |
| **Stiffness** | |
| 1) Morning stiffness | 0 1 2 3 4 |
| 2) Stiffness occurring later in the day | 0 1 2 3 4 |
| **Physical Function** | |
| 1) Descending stairs | 0 1 2 3 4 |
| 2) Ascending stairs | 0 1 2 3 4 |
| 3) Rising from sitting | 0 1 2 3 4 |
| 4) Standing | 0 1 2 3 4 |
| 5) Bending to floor | 0 1 2 3 4 |
| 6) Walking on flat surface | 0 1 2 3 4 |
| 7) Getting in / out of car | 0 1 2 3 4 |
| 8) Going shopping | 0 1 2 3 4 |
| 9) Putting on socks | 0 1 2 3 4 |
| 10) Lying in bed | 0 1 2 3 4 |

| | |
|---|---|
| 11) Taking off socks | 0 1 2 3 4 |
| 12) Rising from bed | 0 1 2 3 4 |
| 13) Getting in/out of bath | 0 1 2 3 4 |
| 14) Sitting | 0 1 2 3 4 |
| 15) Getting on/off toilet | 0 1 2 3 4 |
| 16) Heavy domestic duties | 0 1 2 3 4 |
| 17) Light domestic duties | 0 1 2 3 4 |

**Sample size:** Sample size is calculated by using the formula:

**n= (Z×a + Z b)2 [ 1 - p1 + 1 - p2]**

[ ln (1 - e)]2 [ p1 p2] where p1 = 0.333, p2 = 0.442 Error ratio e = 0.4

Type 1 error α=5%

Type II error b = 10% for detecting results with 90% power of study.

Data lost 10%.

Then the sample size comes out to be n=75 in each group = Total 150.

**Data Analysis:** Non-parametric observations (e.g., WOMAC score) were evaluated using the analysis of variance (ANOVA with the Bonferroni correction). Considering possible intra-individual and inter-individual data variations, completion of the entire follow-up period by at least 40 subjects in each group was required. Level of inflammatory markers pre and post administration of turmeric were assessed. The results were analyzed using descriptive statistics and making comparisons among various groups. Discrete (categorical) data were summarized as proportions and percentages (%) and quantitative data were summarized as mean ± SD.

The following statistics were calculated in the present analysis:

**The Chi Square Test**: This test is used for testing the association between variables.

**The Arithmetic Mean:** The most widely used measure of Central tendency is arithmetic mean, usually referred to simply as the mean, calculated as

$$\bar{x} = \Sigma x / n$$

**Where:** $\bar{x}$ = arithmetic mean, $\Sigma x$ = sum of all observations, n = total number of observations.

So arithmetic mean is the sum of all the numbers in the series divided by the count of all numbers in the series

**The Standard deviation (SD)**: It is calculated by using the formula

$$SD = \sqrt{[ (\Sigma x^2 / n) - (\Sigma x)^2 / n^2 ]}$$

**Where:** SD = standard deviation, $\Sigma x^2$ = sum of squared observations, $\Sigma x$ = sum of observations, n = number of observations.

So the standard deviation (SD) is a measure that is used to quantify the amount of variation or dispersion of a set of data values.

**Unpaired t Test:** The independent samples t-test is used when two separate sets of independent and identically distributed samples are obtained, one from each of the two populations being compared.

**ANOVA:** Analysis of Variance (ANOVA) is a parametric statistical technique used to compare datasets. It is a statistical method used to test differences between two or more means. In this technique inferences about means are made by analyzing variance.

**Univariate block Analysis:** Univariate involves the analysis of a single dependent variable on which influences of various categorical variables and their interactions are observed.

**Results**

**Table 1: Intergroup Comparison of Pain & Physical Activity (WOMAC Score) between Treatment (Group-I) and Placebo (Group-II) Group**

| Group1 | Group – I | | Group – II | | t-value | p-value |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | |
| WOMAC SCORE 1st | 45.08 | 11.56 | 48.92 | 12.28 | -1.97 | 0.051 |
| WOMAC SCORE 2nd | 24.64 | 18.34 | 43.88 | 14.17 | -7.19 | <0.001 |
| diff WOMAC | -20.44 | 14.11 | -5.04 | 8.26 | -8.15 | <0.001 |

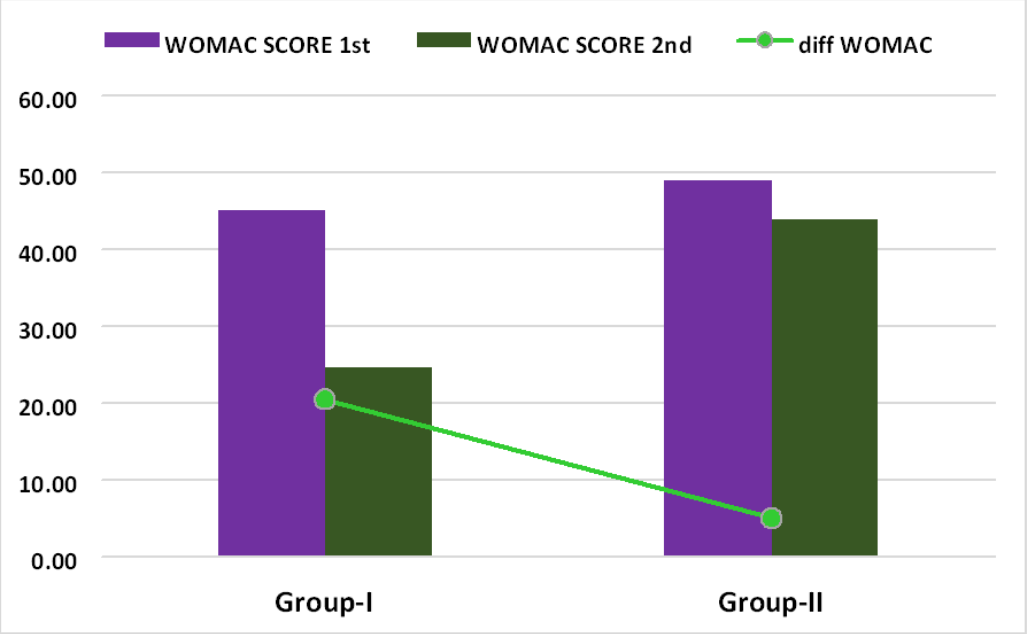


**Figure 1: Intergroup Comparison of Pain & Physical Activity (WOMAC)**

**Score) between Treatment (Group-I) and Placebo (Group-II) Group:** On comparing the WOMAC scores between Group-I and Group-II at first visit, it was found that the mean WOMAC score of Group-I was 45.08±11.56 while the mean WOMAC score of Group II was 48.92±12.28. The difference in mean WOMAC score between Group I and Group II was not found to be significant (p=0.051). On comparing the WOMAC scores between Group-I and Group-II at last visit after 4 months, it was found that the mean WOMAC score of Group-I was 24.64±18.34 while the mean WOMAC score of Group II was 43.88±14.17. The difference in mean WOMAC score between Group I and Group II was found to be statistically significant (p<0.001). On comparing the WOMAC scores change from 1st visit to last visit (after 4 months) between Group-I and Group-II, it was found that the mean WOMAC score change of Group-I was 20.44±14.11 while the mean WOMAC score change of Group II was 5.04±8.26. The mean WOMAC score change in Group I was found to be significantly more than the change in Group II (p<0.001).

**Table 2: Intergroup Comparison of Inflammatory Marker hsCRP between Treatment (Group-I) and Placebo (Group-II) Group**

| Group1 | Group – I | | Group - II | | t-value | p-value |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | |
| hsCRP 1st | 14.24 | 4.77 | 15.31 | 3.49 | -1.58 | 0.117 |
| hsCRP 2nd | 9.82 | 4.27 | 15.75 | 3.61 | -9.18 | <0.001 |
| diff hsCRP | -4.42 | 3.94 | 0.43 | 1.29 | -10.13 | <0.001 |

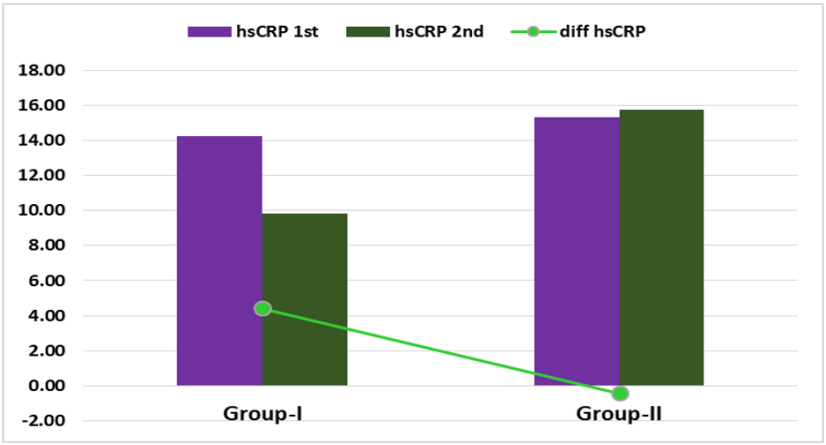


**Figure 2: Intergroup Comparison of Inflammatory Marker hsCRP between Treatment (Group-I) and Placebo (Group-II) Group**

On comparing the hsCRP level between Group-I and Group-II at first visit, it was found that the mean hsCRP level of Group-I was 14.24±4.77 while the mean hsCRP level of Group II was 15.31±3.49. The difference in mean hsCRP level between Group I and Group II was not found to be significant at first visit (p=0.117).

On comparing the hsCRP level between Group-I and Group-II at last visit (after 4 months), it was found that the mean hsCRP level of Group-I was 9.82±4.27 while the mean hsCRP level of Group II was 15.75±3.61. The difference in mean hsCRP level between Group I and Group II was found to be statistically significant at second visit (p<0.001).

On comparing the hsCRP level change from 1st visit to last visit (after 4 months) between Group-I and Group-II, it was found that the mean hsCRP level change of Group-I was 4.42±3.94 while the mean hsCRP level change of Group II was 0.43±1.29. The mean hsCRP level change in Group I was found to be significantly more than the Group II (p<0.001).

**Table 3: Intergroup Comparison of Inflammatory Marker IL-6 between Treatment (Group-I) and Placebo (Group-II) Group**

| Group1 | Group – I | | Group - II | | t-value | p-value |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | |
| IL-6 1st | 20.23 | 13.01 | 22.61 | 15.25 | -1.03 | 0.304 |
| IL6- 2nd | 14.12 | 8.35 | 22.97 | 14.89 | -4.49 | <0.001 |
| diff IL6 | -6.10 | 8.84 | 0.35 | 2.54 | -6.08 | <0.001 |

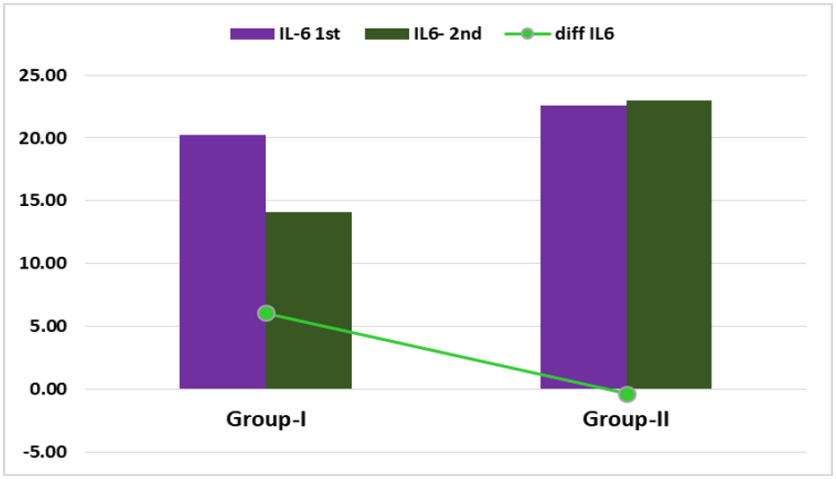


**Figure 3: Intergroup Comparison of Inflammatory Marker IL-6 between Treatment (Group-I) and Placebo (Group-II) Group**

On comparing the IL-6 level between Group-I and Group-II at first visit, it was found that the mean IL-6 level of Group-I was 20.23±13.01 while the mean IL-6 level of Group II was 22.61±15.25. The difference in mean IL-6 level between Group I and Group II was not found to be significant at first visit (p=0.304).On comparing the IL-6 level between Group-I and Group-II at last visit (after 4 months), it was found that the mean IL-6 level of Group-I was 14.12±8.35 while the mean IL-6 level of Group II was 22.97±14.89. The difference in mean IL-6 level between Group I and Group II was found to be statistically significant at second visit (p<0.001). On comparing the IL-6 level change from 1st visit to last visit (after 4 months) between Group-I and Group-II, it was found that the mean IL-6 level change of Group-I was 6.10±8.84 while the mean IL-6 level change of Group II was 0.35±2.54. The mean IL-6 level change in Group I was found to be significantly more than the Group II (p<0.001).

**Table 4: Multivariate Analysis Showing Effect of Various Parameters and Treatment on Pain/Physical activity and Inflammatory Marker Changes**

| Source | | F | p-value | Partial Eta Squared (effect size) |
|---|---|---|---|---|
| Corrected Model | diff WOMAC | 9.092 | <0.001 | 0.340 |
| | diff hsCRP | 12.954 | <0.001 | 0.424 |
| | diff IL6 | 5.168 | <0.001 | 0.227 |
| Intercept | diff WOMAC | 15.929 | <0.001 | 0.102 |
| | diff hsCRP | 4.625 | 0.033 | 0.032 |
| | diff IL6 | 3.589 | 0.060 | 0.025 |
| Age | diff WOMAC | 3.856 | 0.052 | 0.027 |
| | diff hsCRP | 1.205 | 0.274 | 0.008 |
| | diff IL6 | 1.654 | 0.201 | 0.012 |
| Group | diff WOMAC | 34.350 | <0.001 | 0.196 |
| | diff hsCRP | 40.022 | <0.001 | 0.221 |
| | diff IL6 | 11.580 | 0.001 | 0.076 |
| Sex | diff WOMAC | 0.219 | 0.641 | 0.002 |
| | diff hsCRP | 0.114 | 0.736 | 0.001 |
| | diff IL6 | 0.117 | 0.733 | 0.001 |
| METSYN | diff WOMAC | 0.299 | 0.585 | 0.002 |
| | diff hsCRP | 0.235 | 0.629 | 0.002 |
| | diff IL6 | 0.926 | 0.337 | 0.007 |
| Group1 * sex | diff WOMAC | 0.131 | 0.718 | 0.001 |
| | diff hsCRP | 0.154 | 0.695 | 0.001 |
| | diff IL6 | 0.017 | 0.895 | 0.000 |
| Group1 * METSYN | diff WOMAC | 0.157 | 0.693 | 0.001 |
| | diff hsCRP | 0.004 | 0.949 | 0.000 |
| | diff IL6 | 0.716 | 0.399 | 0.005 |
| sex * METSYN | diff WOMAC | 0.417 | 0.520 | 0.003 |
| | diff hsCRP | 0.048 | 0.827 | 0.000 |
| | diff IL6 | 0.032 | 0.858 | 0.000 |
| Group1 * sex * METSYN | diff WOMAC | 0.205 | 0.652 | 0.001 |
| | diff hsCRP | 0.114 | 0.736 | 0.001 |
| | diff IL6 | 0.030 | 0.863 | 0.000 |

The multivariate analysis for searching effects of various parameters and Treatment on Pain/Physical activity and Inflammatory Marker Changes revealed that, On WOMAC score change, maximum effect was found of the treatment ($p<0.001$, effect size=0.196).

None of the other parameters affected WOMAC score significantly ($p>0.05$). On hsCRP level change, maximum effect was found of the treatment ($p<0.001$, effect size=0.221). None of the other parameters affected hsCRP level significantly ($p>0.05$). On IL-6 level change, maximum effect was found of the treatment ($p=0.001$, effect size=0.076). None of the other parameters affected IL-6 level significantly ($p>0.05$).

**Discussion**

In our study it was observed that comparing the WOMAC scores between Group-I and Group-II at first visit, the difference in mean WOMAC score between Group I and Group II was found to be insignificant ($p=0.051$)

On comparing the WOMAC scores between Group-I and Group-II at last visit, the difference in mean WOMAC score between Group I and Group II was again found to be statistically significant ($p<0.001$).

On comparing the WOMAC scores change from 1st visit to last visit between Group-I and Group-II, it was observed that The mean WOMAC score change in Group I was found to be significantly more than the change in Group II ($p<0.001$).

This can be attributed to the fact that the first group was receiving turmeric capsules whereas second group had been given placebo.

Or study was in accordance to the results of the studies by Karlapudi V et al[9], Haroyan A et al[10], Perkins K et al[11] and Ha L et al[12], where it was observed that Curcumin and its components helped in reduction of WOMAC score significantly with use over time.

Haroyan A et al [10] observed Favorable effects of Curcumin compared to placebo after only 3 months of continuous treatment. A significant effect of Curamin® compared to placebo was observed both in physical performance tests and the WOMAC joint pain index. The effect size compared to

placebo was comparable for both treatment groups but was superior in the Curamin® group.

Perkins K et al[11] observed that treatment with curcumin compounds was well tolerated. When compared with active control, curcuma-containing products were similar to nonsteroidal anti-inflammatory drugs, and potentially to glucosamine.

In our study, on comparing the hsCRP level between Group-I and Group-II at first visit, it was not found to be significant. (p=0.117) However, on comparing the hsCRP level between Group-I and Group-II at last visit the difference in mean hsCRP level between Group I and Group II was found to be statistically significant (p<0.001). On comparing the hsCRP level change from 1st visit to last visit between Group-I and Group-II, it was found that mean hsCRP level change in Group I was found to be significantly more than the Group II (p<0.001).

On comparing the IL-6 level between Group-I and Group-II at first visit, The difference in mean IL-6 level between Group I and Group II was not found to be significant (p=0.304). However on comparing the IL-6 level between Group-I and Group-II at last visit, it was observed that the difference in mean IL-6 level between Group I and Group II was found to be statistically significant at second visit (p<0.001).

On comparing the IL-6 level change from 1st visit to last visit between Group-I and Group-II, it was found that the mean IL-6 level change in Group I was found to be significantly more than the Group II (p<0.001).

All the above findings are in accordance to the studies of Akuri MC et ali [13], Sun Y et al [14], Panahi Y et al [15] and Srivastava S et al[16], who in their respective studies observed fall in oxidative stress in patients of OA receiving curcumin therapy vs. those who were only on NSAIDS or other drugs. Thus curcumin has a definitive role in reducing inflammation in OA patients. Akuri MC et al [13] observed that curcumin may play an important role as anti-inflammatory, down-regulating enzymes as phospholipase A2, cyclooxygenase-2, and lipoxygenases, and reducing tumor necrosis factoralpha-and interleukins such as interleukin-1β (IL-1β), IL-6, and IL-8. They also act as inducer of apoptosis in synoviocytes, decreasing the inflammation process and may also reduce the synthesis of reactive oxygen species.

Similar role was also seen in the study by Sun Y et al where administration of curcumin significantly reduced osteoarthritis disease progression in DMM model of osteoarthritis. Curcumin suppressed mRNA expression of pro-inflammatory mediators in arthrodial cartilage of mice subjected to surgery. In LPS- and ATP-induced THP-1 macrophage cells, curcumin significantly suppressed the expression of interleukin 1 beta (IL-1β) and tumor necrosis factor alpha (TNF-α) at both RNA and protein levels. Results from the study of Srivastava S et al showed significant improvement in the patients of curcumin Longa extract group as compared to placebo group. Clinically, the VAS and WOMAC scores became better, and simultaneously, the levels of biomarkers, viz., IL-1β, ROS, and MDA, were also significantly ($p < 0.05$) improved. At the same time, CL decreases the oxidative stress also.

Thus it was seen in our study that therapy with Curcumin Longa extract helps in reducing oxidative stress as well as reduction in biomarkers of inflammatory process associated with osteoarthritis. Turmeric provides improvement in WOMAC score and reduction in inflammatory markers hsCRP and IL-6, establishing the role of curcumin in the treatment of OA.

## Conclusion

To conclude OA apart from being a degenerative disease, may also be a pro- inflammatory state. The greater amount of pain relief in OA patients who were given turmeric makes it a viable alternative to allopathic medicines. Turmeric is already part of our daily diet and its healing properties remain underutilised in treating OA. Significant reduction in inflammatory markers in OA patients after turmeric administration suggests its anti-inflammatory property may have a significant role in symptom relief in OA patients. Time has come to tap the potential health benefits of turmeric on a large scale.

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