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Efficacy and Safety of Amrith Noni Artho Plus in Gout Flare: A Double-Blind, Randomized Clinical Study

Sourabh Giri Baballi Umeshgiri[1], Apoorva Nagraj[1], Radhika Hollehonnuru Meghraj[1], Anand Kumar Upadhyay[2], A. K. Srinivasamurthy[1]

[1]Valyou Products Private Limited, Ramenakoppa, Karnataka – 577226, India.
[1]Valyou Products Private Limited, Ramenakoppa, Karnataka – 577226, India.
[1]Valyou Products Private Limited, Ramenakoppa, Karnataka – 577226, India.
[2]Department of Orthopedics, Rambilas Hospital, Varanasi, Uttar Pradesh 221106
[1]Valyou Products Private Limited, Ramenakoppa, Karnataka – 577226, India

Corresponding Author: Dr. A. K. Srinivasamurthy

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ABSTRACT

Background and Objective: Gout, a metabolic disorder characterized by hyperuricemia and joint inflammation, is a growing global health concern. This study aimed to evaluate the efficacy and safety of Amrith Noni Artho Plus, an Ayurvedic formulation, in managing gout flare.
Study Design: This was a double-blind, randomized, placebo-controlled clinical study conducted over 24 weeks. Forty participants with gout flare were randomized into two groups: the test group received Amrith Noni Artho Plus, while the placebo group received a matched placebo.
Methods: Participants were selected based on inclusion and exclusion criteria. The study was conducted in compliance with the Declaration of Helsinki and ICH-GCP guidelines. Efficacy was assessed through changes in pain intensity (Visual Analogue Scale, VAS), serum uric acid levels, high-sensitivity C-reactive protein (hs-CRP), health assessment questionnaire (HAQ) score, and health-related quality of life (HRQOL, SF-36 questionnaire). Safety was evaluated through adverse events and laboratory parameters. Data were analyzed using descriptive statistics, t-tests, and ANCOVA.
Results: The test group showed significant reductions in pain intensity (-41.50%, $p < 0.0001$), hs-CRP (-28.40%, $p < 0.0001$), and serum uric acid levels (-27.89%, $p < 0.0001$) compared to the placebo group. HAQ scores improved significantly in the test group (-30.41%, $p < 0.0001$), indicating better functional ability. HRQOL scores improved significantly in the test group across all domains ($p < 0.0001$). No adverse events were reported, and all safety parameters remained within normal ranges.
Conclusion: Amrith Noni Artho Plus is an effective and safe intervention for managing gout flare, offering significant improvements in pain, inflammation, uric acid levels, and quality of life. Its favourable safety profile makes it a promising alternative to conventional gout treatments.

Keywords: Gout flare, Amrith Noni Artho Plus, Randomized Clinical Study, hyperuricemia, anti-inflammatory.

INTRODUCTION

Gout, a metabolic disorder caused by excessive uric acid accumulation in joints, is a growing global health concern. It results from purine metabolism dysfunction, leading to symptoms such as pain, swelling, and joint stiffness[1]. In chronic cases, it can cause joint deformities, gout nodules, and complications such as kidney disease or uric acid stones[2]. The pathophysiology of gout involves interactions between monosodium urate crystals and tissues during purine catabolism, where the enzyme xanthine oxidase converts purines to uric acid, leading to hyperuricemia and crystal deposition in joints[3,4]. Humans lack the enzyme uricase, which converts uric acid to the more soluble allantoin, making them susceptible to hyperuricemia[5].

Several risk factors contribute to the development of gout, including hyperuricemia, age, genetic, environmental factors, dietary habits, alcohol consumption, metabolic syndrome, hypertension, obesity, diuretic use, cholesterol levels, and chronic renal disease[6]. Gout is no longer limited to older populations, as its onset is increasingly observed in younger population[6]. Men are at a higher risk of developing gout compared to women, although the risk in women increases during post-menopause due to the loss of the uricosuric action of estrogen[7]. According to the World Health Organization (2019), the global prevalence of gout is 3.9%, with its incidence rising annually[8]. It is estimated that the global prevalence of gout will increase by more than 70% from 2020 to 2050[9].

The management of gout primarily focuses on alleviating the severe pain and inflammation associated with acute attacks. Conventional treatments, including NSAIDs, colchicine, glucocorticoids, and allopurinol, provide short-term relief but are associated with significant side effects, such as skin allergies, fever, diarrhoea, renal dysfunction, myocardial infarction, and hepatic dysfunction[10,11]. These adverse effects often lead to discontinuation of treatment, complicating gout management and necessitating the exploration of alternative therapies, particularly those derived from medicinal plants[12].

Traditional systems of medicine, such as Ayurveda, offer a holistic approach to gout management, focusing on balancing the body's doshas (Vata, Pitta, and Kapha) and addressing the root cause of the disease. Ayurvedic treatments often involve herbal formulations, dietary modifications, and lifestyle changes to reduce inflammation, lower uric acid levels, and prevent recurrent gout flares[13]. Amrith Noni Artho Plus, an Ayurvedic formulation, contains *Morinda citrifolia* (Noni) as its major ingredient, along with other herbs. Noni has been traditionally used for its analgesic, anti-inflammatory, and antioxidant properties, which may help alleviate the symptoms of gout and reduce uric acid levels[14]. This double-blind, randomized clinical study aimed to evaluate the efficacy and safety of Amrith Noni Artho Plus in the treatment of gout flares.

METHODS

Study Design

This study was a double-blinded, randomized, placebo-controlled clinical trial designed to evaluate the efficacy and safety of Amrith Noni Artho Plus in patients with gout flare over a 24-week period. The study consisted of four scheduled visits at screening (day -1 to -2), baseline (day 0), Week 12, and Week 24. Participants were randomized into either the intervention or placebo group. Efficacy was assessed through the changes in gout flare severity and frequency, while safety was monitored via adverse events and laboratory safety parameters.

Study Population and Sample Size

Male and female subjects aged 18 years and older, diagnosed with gout flare, were enrolled in the study. A total of 40 eligible participants were recruited and randomized into two groups-the placebo group and the test group-in a 1:1 ratio. The sample size of 40 subjects (20 per group) was determined based on practical considerations rather than

statistical calculations, ensuring feasibility while maintaining a balanced distribution for comparative analysis.

Selection of Participants

Inclusion criteria

The study enrolled male and female participants aged 18 years and older with gout flare. Eligible participants had a confirmed diagnosis of chronic gout by a general practitioner or orthopaedic physician, with or without supporting diagnostic tests such as joint aspiration, blood tests, or imaging. Individuals who had no plans to initiate new treatments during the study period, did not regularly use anti-inflammatory or analgesic medications, or were dissatisfied with their current treatment regimen were included.

Exclusion criteria

Participants were excluded if they had glucose-6-phosphate dehydrogenase deficiency, a history of surgery, gastrointestinal bleeding, or gastric ulcers. Those with rheumatoid arthritis, osteoarthritis, or septic arthritis within the past 3 months, or uncontrolled medical conditions such as cardiovascular diseases, impaired liver or kidney function, malignancies, or psychiatric disorders, were also excluded. Other exclusion criteria included the use of complementary and alternative medicines, addiction to psychotropic agents or opioids, prior use of uricase-containing drugs, anticoagulants, NSAIDs, or low-dose colchicine, and any history of allergy to the investigational product. Pregnant or lactating women, women of childbearing age not using adequate contraception, and those who participated in another clinical trial within 30 days of screening were also excluded.

Randomization and Blinding

Eligible subjects were randomly assigned to either the placebo group or the test group using a pre-determined randomization code. The randomization process was conducted in a double-blinded manner, ensuring that neither the participants nor the study personnel were aware of the group assignments.

Test Product

Amrith Noni Artho Plus is a herbal formulation developed to aid in the management of gout and related inflammatory conditions. It contains a blend of medicinal herbs, including Noni fruit juice, Nirgundi, Shallaki, Guggulu, Dashamoola, and others, known for their anti-inflammatory and antioxidant properties. Noni fruit juice, a key ingredient, helps to reduce uric acid levels and alleviate inflammation while also providing essential nutrients such as potassium, vitamin A, and vitamin C, which support cell repair and strengthen the immune system. The combination of these ingredients is intended to improve joint health and overall well-being. In the study, participants in the test group received Amrith Noni Artho Plus, while the control group received a placebo. The dosing regimen was structured to gradually increase over time - starting with 5 ml twice daily mixed with lukewarm water during the first week, increasing to 10 ml in the second week, and 15 ml from the third week onward. All doses were administered on an empty stomach, 30 minutes before meals.

Outcome measures

The primary outcome of the study was the change in the Visual Analogue Scale (VAS) score from baseline to the end of the 24-week treatment period to assess the reduction in pain intensity associated with gout flare. Secondary outcomes included changes in serum uric acid levels, high-sensitivity C-reactive protein (hs-CRP) levels, and Health Assessment Questionnaire (HAQ) pain scores, all measured from baseline to the end of treatment. Additionally, health-related quality of life was assessed using the SF-36 questionnaire, while patient and physician global evaluations provided further insights into treatment effectiveness. Radiological changes were monitored via X-ray, and vital

signs were recorded to track physiological responses. Laboratory safety parameters were analyzed to ensure treatment safety, and the incidence of adverse events was documented to evaluate tolerability.

Ethics and informed consent

The study protocol was approved by the Vatsalya Ethics Committee (IEC), Varanasi, Uttar Pradesh, India on August 8, 2023. The study was conducted in compliance with the Declaration of Helsinki, ICMR Ethical Guidelines for Biomedical Research (2006), ICH-GCP, and ASU-GCP. The informed consent process was carefully conducted to ensure participants fully understood the objectives, procedures, potential risks, and benefits of the study. Written consent was obtained only after participants were provided with sufficient time to carefully consider their decision to participate. The study is registered with the Clinical Trials Registry - India (CTRI) (Number: CTRI/2023/10/058982) dated October 4, 2023.

Data collection, and compliance

Data collection and compliance were conducted in accordance with GCP guidelines, applicable regulations, and sponsor-defined procedures. The study monitor maintained regular contact with the study site through periodic on-site visits and remote communications. Monitoring activities included assessing study progress, verifying data accuracy and completeness, and performing source document verification by comparing Case Report Forms (CRFs) with source documents such as medical records and consent forms. Any identified issues were addressed promptly to maintain data integrity and subject safety. The investigator ensured transparency and compliance by providing the monitor with direct access to all relevant documents and facilitating discussions on findings and concerns. Before the study began, the monitor reviewed the protocol and data collection processes with site personnel to ensure alignment with study requirements. All necessary closure activities were completed in accordance with the protocol guidelines.

STATISTICAL METHODS

The analysis was conducted on a per-protocol (PP) basis. Descriptive statistics were used to describe the study population, with data summarized and presented as numbers, percentages, means, and standard deviations (SD). Efficacy and safety variables were analyzed using descriptive statistics, including arithmetic means, SDs, percentages for quantitative variables, and frequencies for qualitative variables. Within-group comparisons from baseline to different visits were performed using a t-test, while inter-group comparisons were conducted using Analysis of Covariance (ANCOVA). A p-value of less than 0.05 was considered statistically significant. Statistical analysis was performed using SPSS software (Version 10.0).

RESULTS

Baseline and Demographic Data

The study initially aimed to enrol 40 subjects, and of the 42 screened subjects, 2 were screen failures and 40 eligible subjects were enrolled in this study. Among the 40 enrolled subjects, 57.9% females in the placebo group and 42.1% in the test group. The mean age of the subjects in the placebo and test groups were 47.55 ± 12.61 and 46.25 ± 13.01 years, respectively. Similarly, mean height (Placebo: 159.90 ± 5.54 cm; Test: 161.55 ± 5.81 cm; P = 0.36), weight (Placebo: 61.13 ± 6.56 kg; Test: 61.10 ± 5.71 kg; p = 0.99), and BMI (Placebo: 23.84 ± 1.30 kg/m²; Test: 23.36 ± 0.98 kg/m²; p = 0.20) did not differ significantly between groups (Table 1).

Table 1: Summary of baseline and demographic data

Variables	Placebo Group (n=20)	Test Group (n=20)	p value
Female, n(%)	11(57.90)	8(42.10)	0.34^
Male, n(%)	9(42.90)	12(57.10)	
Age (yrs), mean ± sd	47.55 ± 12.605	46.25 ± 13.01	0.75$
Height (cm), mean ± sd	159.9 ± 5.543	161.55 ± 5.808	0.36$
Weight (kg), mean ± sd	61.13 ± 6.561	61.1 ± 5.709	0.99$
BMI (kg/m^2), mean ± sd	23.84 ± 1.295	23.36 ± 0.983	0.20$

$p: Between groups comparison analysed using independent t-test and ^p: Analysed using chi-square test.

Efficacy Outcomes

Body weight and BMI

Table 2 outlines the changes in body weight and BMI across assessment visits. At baseline (day 0), the placebo group had a mean weight of 60.74 ± 6.49 kg compared to 61.15 ± 5.68 kg for the test group. At week 12, mean weight in the test group increased to 61.77 ± 5.37 kg, indicating 1.01% change, while the placebo group showed minimal increase to 60.90 ± 7.20 kg. At week 24, mean weight in the test group and placebo group was significantly increased to 62.19 ± 5.25 kg (p = 0.002), and 61.50 ± 6.88 kg (p = 0.007), respectively, but between group differences were not significant (p = 0.480). Similarly, BMI changes followed a comparable trend. At baseline (day 0), the placebo group had mean BMI of 23.81 ± 1.30 kg/m² versus 23.38 ± 0.97 kg/m² for the test group. At visit 4, the mean BMI in the test group significantly increased to 23.79 ± 0.95 kg/m² (1.75%, p = 0.002), while in the placebo group increased to 24.11 kg/m² ± 1.44 (1.26% change, p = 0.008).

Table 2: Body weight and BMI at different assessment visits.

Variables	Placebo group (n=19)	Test group (n=20)	$p value
Weight (kg)			
Day 0 (mean ± sd)	60.74 ± 6.49	61.15 ± 5.68	-
Week 12 (mean ± sd)	60.90 ± 7.20	61.77 ± 5.37	0.20
%change	0.26%	1.01%	-
*p-value	0.567	0.006	
Week 24 (mean ± sd)	61.5 ± 6.88	62.19 ± 5.25	0.48
%change	1.25%	1.70%	-
*p-value	0.007	0.002	
BMI (kg/m^2)			
Day 0 (mean ± sd)	23.81 ± 1.30	23.38 ± 0.97	-
Week 12 (mean ± sd)	23.86 ± 1.51	23.63 ± 0.99	0.11
%change	0.21%	1.07%	-
*p-value	0.678	0.004	
Week 24 (mean ± sd)	24.11 ± 1.44	23.79 ± 0.95	0.51
%change	1.26%	1.75%	-
*p-value	0.008	0.002	

***p: Within group comparison analysed using paired t test and $p: Between groups comparison analysed using ANCOVA**

Pain intensity

The analysis of pain intensity revealed significant differences between the placebo and test groups over 24 weeks (Table 3). At baseline (day 0), pain intensity was comparable between the placebo group (58.49 ± 9.23) and the test group (61.2 ± 9.73). At week 12, the test group demonstrated a significant reduction in pain intensity (47.16 ± 10.30), reflecting 22.94% decrease (p < 0.0001), while the placebo group showed negligible change (-0.74%, p = 0.510). At week 24, the test group showed further reduction in pain intensity to 35.8 ± 9.87 (-41.50%, p < 0.0001), whereas the placebo group showed modest reduction to 54.17 ± 7.95 (-7.39%, p < 0.0001). Between-group comparisons at both week 12 and week 24, pain intensity in the test group showed significant reduction (p < 0.0001) compared to placebo group (Figure 1).

Table 3: Within groups differences in pain intensity at different assessment points

	Placebo Group (n=19)	Test Group (n=20)
Day 0 (mean ± sd)	58.49 ± 9.23	61.2 ± 9.73
Week 12 (mean ± sd)	58.06 ± 8.36	47.16 ± 10.30
%change	-0.74%	-22.94%
*p-value	0.510	<0.0001
Week 24 (mean ± sd)	54.17 ± 7.95	35.8 ± 9.87
%change	-7.39%	-41.50%
*p-value	<0.0001	<0.0001

[*]p: Within group comparison analysed using paired t test. Day 0 vs week 12 and week 24.

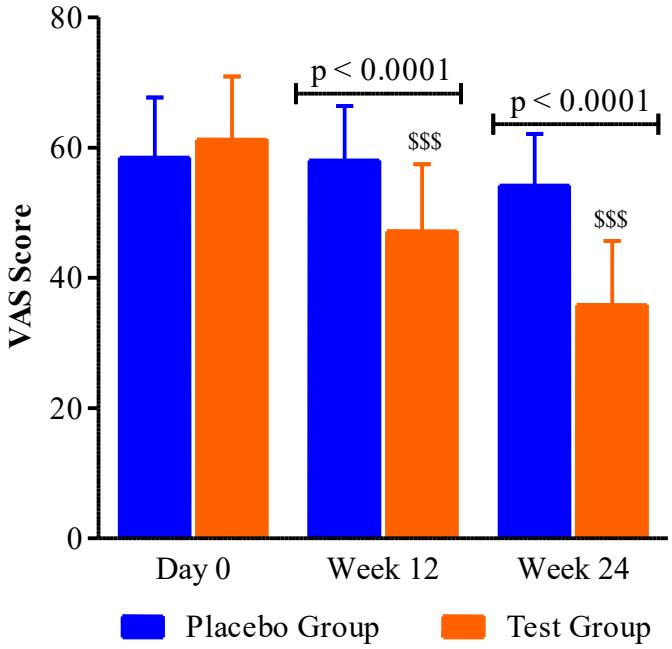


Figure 1: Between groups comparison in pain intensity at different assessment points. Placebo group vs test group comparisons were performed using ANCOVA. [$$$]p <0.0001.

Physician and subjective global assessment (PGA and SGA)

Both the placebo and test groups showed reductions in PGA and SGA scores over time, with significantly greater improvements in the test group (Table 4). In the test group, PGA scores decreased from 5.15 ± 0.59 at baseline to 2.90 ± 0.72 at week 12 (-43.69%, $p < 0.0001$) and further to 1.35 ± 0.49 at week 24 (-73.79%, $p < 0.0001$). The placebo group experienced a smaller reduction from 5 ± 0.47 at baseline to 4.37 ± 0.68 at week 12 (-12.60%, $p = 0.004$) and 4.21 ± 0.71 at week 24 (-15.80%, $p < 0.0001$). Similarly, SGA scores in the test group dropped significantly from 5.6 ± 0.50 at baseline to 3.4 ± 0.60 at week 12 (-39.29%, $p < 0.0001$) and further to 1.8 ± 0.70 at week 24 (-67.86%, $p < 0.0001$). The placebo group showed a reduction from 5.53 ± 0.51 at baseline to 3.42 ± 0.51 at week 12 (-38.16%, $p < 0.0001$) and 3.37 ± 0.50 at week 24 (-39.06%, $p < 0.0001$).

Between-group comparisons showed significantly greater improvements in the test group compared to the placebo group (Figure 2). At week 12, the test group had a significantly ($p < 0.0001$) lower PGA score (2.90 ± 0.72) than the placebo group (4.37 ± 0.68). This difference was even more pronounced at week 24, where the PGA score in the test group (1.35 ± 0.49) was significantly lower than the placebo group ($p < 0.0001$). A similar trend was observed in SGA scores, where there was no significant difference between groups at week 12, but at the week 24, the test group had significantly

lower scores (1.8 ± 0.70, p < 0.0001) compared to the placebo group (3.37 ± 0.50).

Table 4: Within the group change in PGA and SGA scores at different assessment points

	Placebo Group (n=19)	Test Group (n=20)
PGA		
Day 0 (mean ± sd)	5 ± 0.47	5.15 ± 0.59
Week 12 (mean ± sd)	4.37 ± 0.68	2.90 ± 0.72
%change	-12.60%	-43.69%
p-value*	0.004	<0.0001
Week 24	4.21 ± 0.71	1.35 ± 0.49
%change (mean ± sd)	-15.80%	-73.79%
p-value*	<0.0001	<0.0001
SGA		
Day 0 (mean ± sd)	5.53 ± 0.51	5.6 ± 0.50
Week 12 (mean ± sd)	3.42 ± 0.51	3.4 ± 0.60
%change	-38.16%	-39.29%
p-value*	<0.0001	<0.0001
Week 24 (mean ± sd)	3.37 ± 0.50	1.8 ± 0.70
%change	-39.06%	-67.86%
p-value*	<0.0001	<0.0001

***p: Within group comparison analysed using paired t test. Day 0 vs week 12 and week 24.**

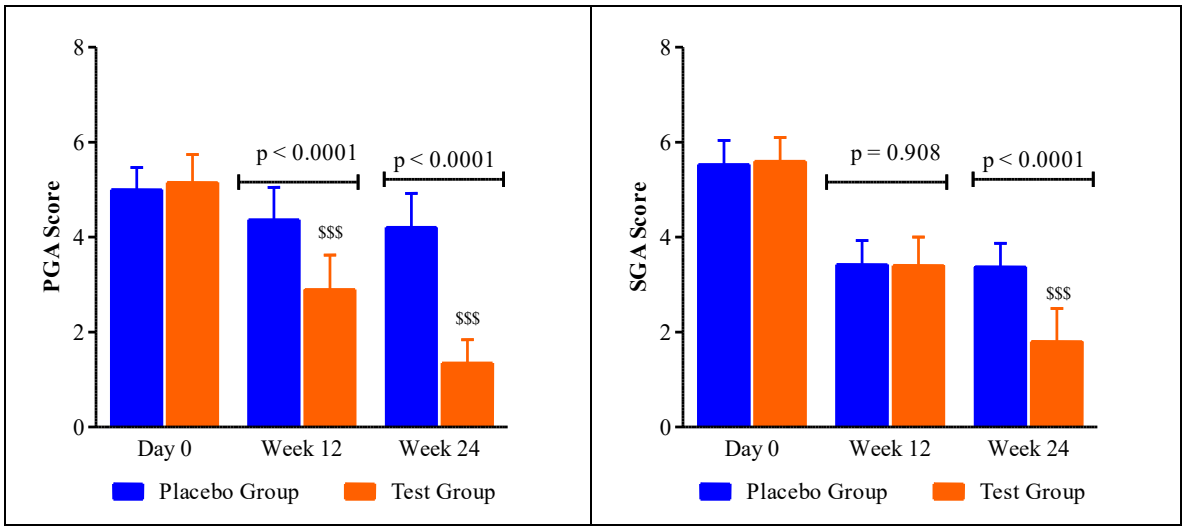


Figure 2: Between groups comparison in PGA and SGA scores at different assessment points. Placebo group vs test group comparisons were performed using ANCOVA. $^{\$\$\$}$p < 0.0001.

Assessment of health

The Health Assessment Questionnaire (HAQ) scores showed greater improvement in the test group compared to the placebo group over the 24-week period (Table 5). Within-group analysis indicated a modest decrease in HAQ scores in the placebo group, from 59.68 ± 6.77 at baseline to 58.37 ± 6.29 at week 12 (-2.20%, p = 0.159) and further to 51.05 ± 7.91 at week 24 (-14.46%, p < 0.0001). In contrast, the test group exhibited a significant reduction in HAQ scores, decreasing from 61 ± 9.06 at baseline to 53.69 ± 8.75 at week 12 (-11.98%, p < 0.0001) and further to 42.45 ± 8.44 at week 24 (-30.41%, p < 0.0001).

Between-group analysis showed a significant difference in HAQ scores between the groups at both week 12 and week 24 (Figure 3). At week 12, the test group had a significantly lower HAQ score (53.69 ± 8.75) compared to the placebo group (58.37 ± 6.29), with p < 0.0001. This difference widened by week 24, where the score in test group (42.45 ± 8.44) was significantly lower than the placebo group (51.05 ± 7.91, p < 0.0001).

Table 5: Within the group change in HAQ scores at different assessment points

	Placebo Group (n=19)	Test Group (n=20)
Day 0 (mean ± sd)	59.68 ± 6.77	61 ± 9.06
Week 12 (mean ± sd)	58.37 ± 6.29	53.69 ± 8.75
%change	-2.20%	-11.98%
*p-value	0.159	<0.0001
Week 24 (mean ± sd)	cc	42.45 ± 8.44
%change	-14.46%	-30.41%
*p-value	<0.0001	<0.0001

***p: Within group comparison analysed using paired t test. Day 0 vs week 12 and week 24.**

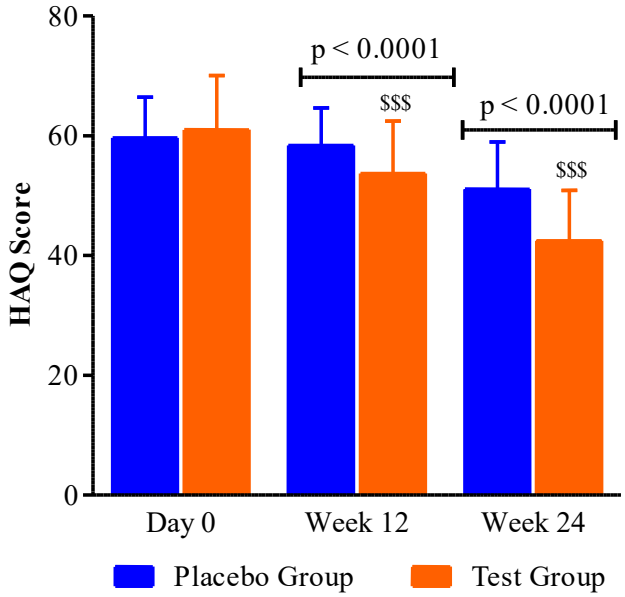


Figure 3: Between groups comparison in HAQ scores at different assessment points. Placebo group vs test group comparisons were performed using ANCOVA. $^{$$$}$p < 0.0001.

Health-related quality of life (HRQOL)

The within-group analysis of HRQOL using SF-36 questionnaire scores showed marked improvements across various domains in the test group compared to the placebo group. In the test group, physical functioning scores increased by 43.65% at week 12 and 100.79% at week 24, while in the placebo group showed minimal improvements of 4.29% and 28.19%, respectively. Physical and emotional role limitations improved by 132% and nearly 100% in the test group at week 24, compared to 28.03% and 31.81% in the placebo group. Vitality scores in the test group improved by 129.17%, while the placebo group showed only 33.59%. Mental health and social functioning also showed greater improvements in the test group (97.30% and 66.66%, respectively) compared to the placebo group (70.90% and 27.0%, respectively). Additionally, body pain and general health perceptions improved significantly in the test group by 82.55% and 93.33%, respectively, whereas the placebo group showed only modest improvements (Table 6).

The between-group analysis revealed statistically significant differences between the test and placebo groups across all domains of the HRQOL. At week 24, the test group had significantly higher physical functioning scores (63.25 ± 7.99) compared to the placebo group (39.47 ± 9.11), with $p < 0.0001$. Physical and emotional role limitations scores showed significantly higher improvements in the test group (132% and 100%, respectively) compared to the placebo group (28.03% and 31.81%, respectively) with $p < 0.0001$. The test group also showed significant improvements in

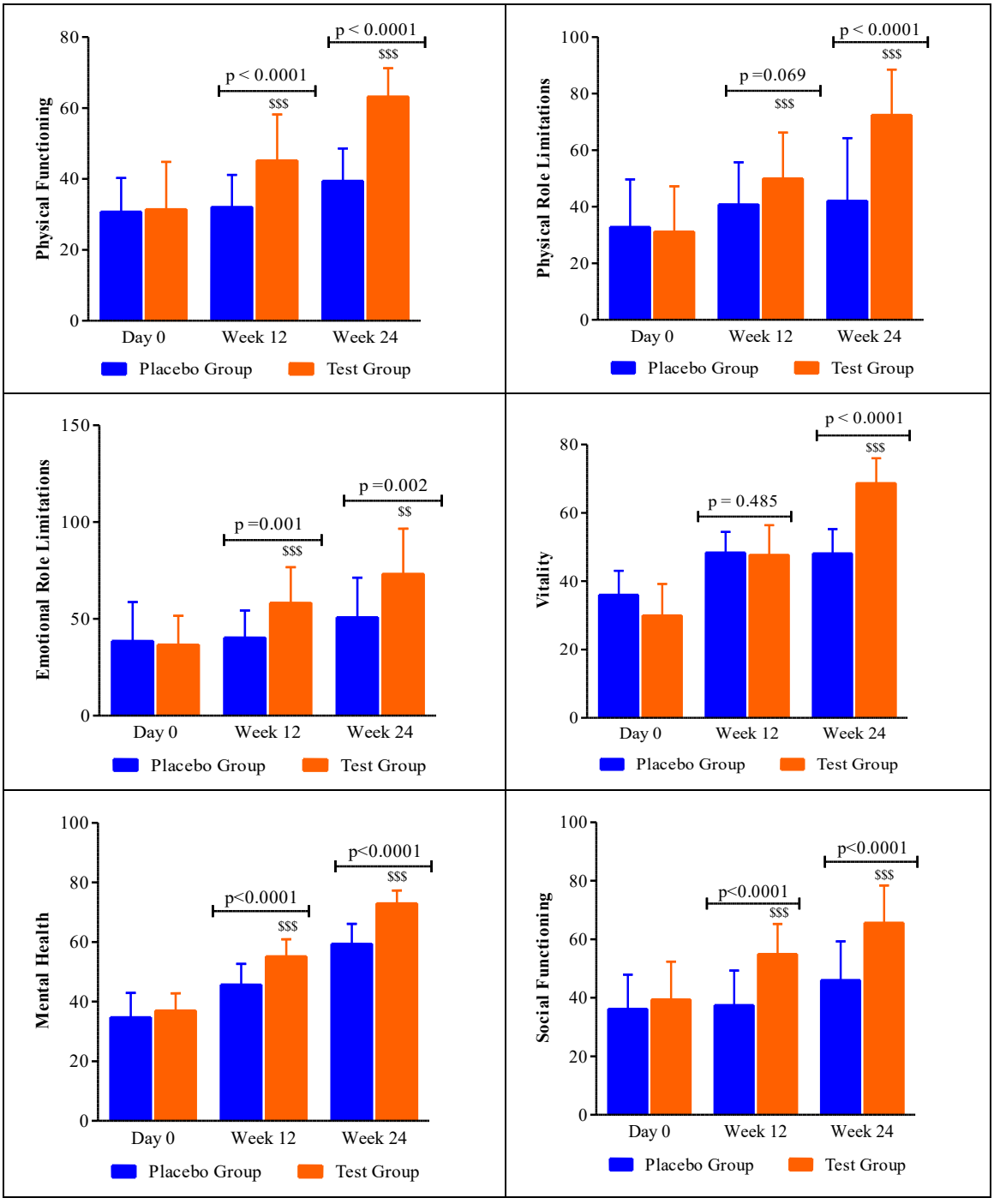
vitality, mental health, social functioning, body pain, and general health perceptions, with all domains showing significant differences ($p < 0.001$).

Table 6: Within group mean change in SF 36 scores of different health dimensions

	Placebo Group (n=19)	Test Group (n=20)
Physical functioning		
Day 0 (mean ± sd)	30.79 ± 9.47	31.5 ± 13.29
Week 12 (mean ± sd)	32.11 ± 9.02	45.25 ± 12.92
%change	4.29%	43.65%
*p-value	0.507	<0.0001
Week 24 (mean ± sd)	39.47 ± 9.11	63.25 ± 7.99
%change	28.19%	100.79%
*p-value	0.004	<0.0001
Physical role limitations		
Day 0 (mean ± sd)	32.89 ± 16.78	31.25 ± 15.97
Week 12 (mean ± sd)	40.82 ± 14.90	50 ± 16.22
%change	24.11%	60.00%
*p-value	0.185	<0.0001
Week 24 (mean ± sd)	42.11 ± 22.13	72.5 ± 16.02
%change	28.03%	132.0%
*p-value	0.130	<0.0001
Emotional role limitations		
Day 0 (mean ± sd)	38.6 ± 20.07	36.67 ± 14.91
Week 12 (mean ± sd)	40.35 ± 13.96	58.33 ± 18.34
%change	4.53%	59.07%
*p-value	0.790	0.002
Week 24 (mean ± sd)	50.88 ± 20.39	73.33 ± 23.20
%change	31.81%	99.97%
*p-value	0.031	<0.0001
Vitality		
Day 0 (mean ± sd)	36.05 ± 6.99	30 ± 9.18
Week 12 (mean ± sd)	48.42 ± 6.02	47.75 ± 8.66
%change	34.31%	59.17%
*p-value	<0.0001	<0.0001
Week 24 (mean ± sd)	48.16 ± 7.11	68.75 ± 7.23
%change	33.59%	129.17%
*p-value	<0.0001	<0.0001
Mental health		
Day 0 (mean ± sd)	34.74 ± 8.12	37 ± 5.79
Week 12 (mean ± sd)	45.68 ± 6.97	55.25 ± 5.65
%change	31.49%	49.32%
*p-value	0.0012	<0.0001
Week 24 (mean ± sd)	59.37 ± 6.70	73 ± 4.28
%change	70.90%	97.30%
*p-value	<0.0001	<0.0001
Social functioning		
Day 0 (mean ± sd)	36.26 ± 11.63	39.38 ± 13
Week 12 (mean ± sd)	37.5 ± 11.79	55 ± 10.26
%change	3.42%	39.66%
*p-value	0.594	<0.0001
Week 24 (mean ± sd)	46.05 ± 13.21	65.63 ± 12.74
%change	27.0%	66.66%
*p-value	0.012	<0.0001
Bodily pain		
Day 0 (mean ± sd)	41.05 ± 13.29	36.5 ± 18.86
Week 12 (mean ± sd)	49.34 ± 5.94	54.38 ± 18.78
%change	20.19%	48.99%

*p-value	0.011	<0.0001
Week 24 (mean ± sd)	53.61 ± 15.91	66.63 ± 14.96
%change	30.60%	82.55%
*p-value	0.002	<0.0001
General health		
Day 0 (mean ± sd)	37.42 ± 5.45	33.75 ± 9.58
Week 12 (mean ± sd)	44.47 ± 9.99	58.5 ± 6.71
%change	18.84%	73.33%
*p-value	0.015	<0.0001
Week 24 (mean ± sd)	48.42 ± 6.25	65.25 ± 6.38
%change	29.40%	93.33%
*p-value	<0.0001	<0.0001

***p: Within group comparison analysed using paired t test. Day 0 vs week 12 and week 24.**



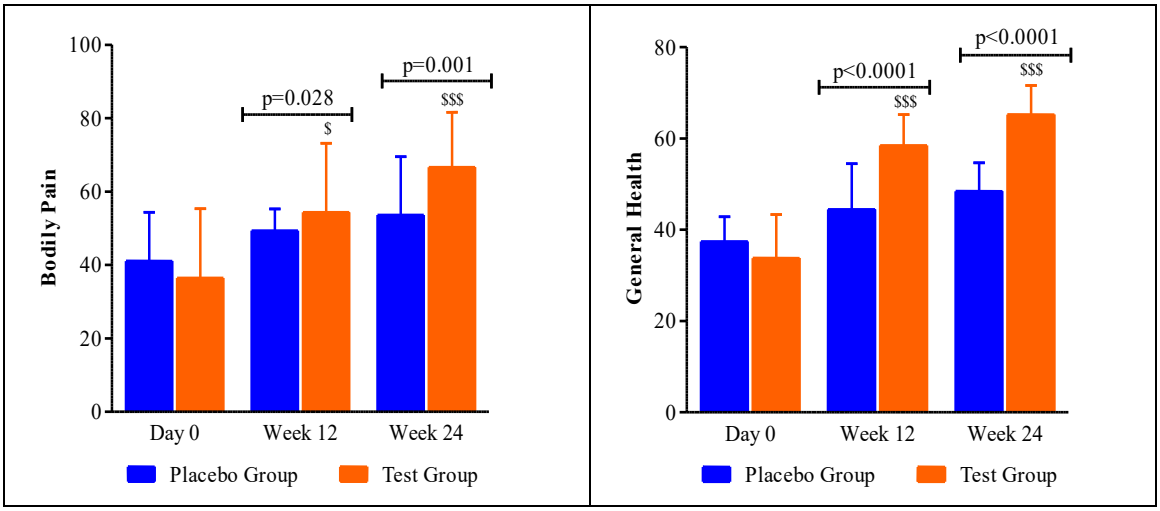


Figure 4: Between groups comparison in SF36 scores of different health dimensions. Placebo group vs test group comparisons were performed using ANCOVA. $p < 0.05, $$p < 0.01 and $$$p < 0.001.

Serum biomarkers

In the test group, hs-CRP levels significantly decreased from 5.07 ± 1.18 mg/L at baseline (day 0) to 4.65 ± 1.11 mg/L at week 12 (8.28% reduction, p<0.0001) and further decreased to 3.63 ± 0.99 mg/L at week 24 (28.40% reduction, p < 0.0001). Uric acid levels dropped from 7.11 ± 1.12 mg/dL at baseline to 6.08 ± 0.85 mg/dL at week 12 (14.37% reduction, <0.0001) and further decreased to 5.12 ± 0.83 mg/dL at week 24 (27.89% reduction, p < 0.0001). In contrast, the placebo group showed a slight decrease in hs-CRP from 5.43 ± 1.16 mg/L to 5.36 ± 1.12 mg/L at week 12 (1.29%) and 5.12 mg/L at week 24 (5.71%). Uric acid levels in the placebo group dropped from 7.28 ± 1.21 mg/dL at baseline to 6.87 ± 1.12 mg/dL at week 12 (5.63%) and 6.62 ± 0.99 mg/dL at week 24 (9.07%). Between-group analysis revealed significant differences in the reduction of both biomarkers (Table 7). The test group showed significantly greater reductions in hs-CRP and uric acid levels compared to the placebo group at both week 12 and week 24 (p < 0.0001), indicating a more pronounced effect of the intervention over time.

Table 7: Within and between groups mean change in serum levels of hs-CRP and uric acid.

	Placebo Group (n=19)	Test Group (n=20)	$p value
hs-CRP (mg/L)			
Day 0 (mean ± sd)	5.43 ± 1.16	5.07 ± 1.18	-
Week 12 (mean ± sd)	5.36 ± 1.12	4.65 ± 1.11	<0.0001
%change	-1.29%	-8.28%	-
*p-value	0.095	<0.0001	
Week 24 (mean ± sd)	5.12 ± 1.19	3.63 ± 0.99	<0.0001
%change	-5.71%	-28.40%	-
*p-value	<0.0001	<0.0001	
Uric Acid (mg/dL)			
Day 0 (mean ± sd)	7.28 ± 1.21	7.1 ± 1.12	-
Week 12 (mean ± sd)	6.87 ± 1.12	6.08 ± 0.85	<0.0001
%change	-5.63%	-14.37%	-
*p-value	0.0002	<0.0001	
Week 24 (mean ± sd)	6.62 ± 0.99	5.12 ± 0.83	<0.0001
%change	-9.07%	-27.89%	-
*p-value	0.0008	<0.0001	

***p: Within group comparison analysed using paired t test and $p: Between groups comparison analysed using ANCOVA.**

Safety and Adverse Events

Vital signs

The analysis of vital signs revealed no significant differences between the test and placebo groups across all visits for pulse rate, blood pressure, and respiratory rate. Pulse rate remained stable, with no statistically significant differences at day 0, week 12, or week 24 ($p > 0.45$). Systolic and diastolic blood pressure values were consistent between groups at all visits, with no between group differences observed. Respiratory rate showed similar trends, with minimal variation between groups and no significant differences noted ($p > 0.46$). Although body temperature showed a slight increase in the test group at week 12 ($p = 0.012$), the change was minimal and not clinically significant.

Routine blood counts

The between-group analysis of routine blood counts showed no significant differences for most hematological parameters between the test and placebo groups across visits. Hemoglobin levels increased slightly more in the test group (2.62%) compared to the placebo group (0.74%), but the difference was not statistically significant. Platelet and total leukocyte count showed minimal changes, with no significant differences between groups. However, RBC counts increased significantly in the test group (3.69%) compared to a slight decrease in the placebo group (-1.31%), with a statistically significant difference ($p = 0.027$). ESR showed a notable reduction of 13.90% in the test group compared to 4.08% in the placebo group, with a highly significant between the group difference ($p < 0.0001$). Other parameters, including neutrophils, lymphocytes, eosinophils, and monocytes, showed no significant differences between groups, and basophil counts remained unchanged in both groups.

Renal and liver functions

The analysis of renal and liver function parameters showed that all parameters remained within normal reference ranges throughout the study. Serum creatinine levels were slightly increased in the Placebo group (4.76%), while decreased in the test group (4.65%), but the levels remained within the normal range. Blood urea nitrogen (BUN) levels showed significant reduction in both groups. Liver function parameters, including serum bilirubin, AST, and ALT, also remained within normal limits, with no significant differences between groups. AST levels decreased by 11.44% in the placebo group and 7.99% in the test group ($p > 0.01$), while ALT levels showed slight variations with no significant between group differences. Serum albumin levels increased slightly in both groups at week 22, but remained within the normal reference range.

Adverse effects

No adverse events were reported or observed among the 39 subjects who completed the study, suggesting that the intervention is safe for clinical use.

DISCUSSION

The present study evaluated the efficacy and safety of Amrith Noni Artho Plus, an Ayurvedic formulation, in managing gout flare over a 24-week period. The results demonstrated significant improvements in pain intensity, serum biomarkers (hs-CRP and uric acid levels), functional ability (HAQ scores), and health-related quality of life (HRQOL) in the test group compared to the placebo group. These findings align with previous pharmacological studies on the anti-inflammatory, analgesic, and uric acid-lowering properties of the key ingredients in Amrith Noni Artho Plus, particularly *M. citrifolia* and other herbal components like Nirgundi, Shallaki, and Guggulu.

The significant reduction in pain intensity observed in the test group, as measured by the VAS, highlights the potent analgesic and anti-inflammatory effects of Amrith Noni Artho Plus. This reduction in pain aligns with the known properties of its key ingredients, such as Noni, Shallaki, and Guggulu, which have been traditionally used to alleviate pain and inflammation. This is consistent with previous studies on Noni, which has been

shown to inhibit cyclooxygenase (COX) and lipoxygenase (LOX) pathways, reducing the production of pro-inflammatory mediators like prostaglandins and leukotrienes[15]. Similarly, Shallaki (*Boswellia serrata*) contains boswellic acids, which have been reported to suppress 5-lipoxygenase activity, thereby reducing inflammation and pain in arthritic conditions[16,17]. The combination of these herbs likely contributed to the observed reduction in pain and inflammation in the test group. The reduction in hs-CRP levels further supports the anti-inflammatory efficacy of the formulation. CRP is a well-established marker of systemic inflammation, and its reduction indicates a decrease in inflammatory activity. This finding aligns with previous research on *C. wightii* and *M. citrifolia*, both of which have been shown to effectively lower CRP levels in patients with chronic inflammatory conditions. The anti-inflammatory properties of these key ingredients in Amrith Noni Artho Plus likely contributed to the observed reduction in CRP levels[18]. The synergistic action of these herbs likely enhanced the overall anti-inflammatory effect of Amrith Noni Artho Plus.

The significant improvement in HAQ scores in the test group indicates enhanced functional ability and reduced disability in patients with gout flare. This improvement is likely due to the combined effects of reduced pain, inflammation, and joint stiffness, which are key contributors to functional limitations in gout patients. The findings are consistent with previous studies on Noni, which has been reported to improve joint mobility and reduce disability in patients with osteoarthritis[19]. The inclusion of Dashamoola, a traditional Ayurvedic formulation known for its musculoskeletal benefits, may have further contributed to the observed improvements in HAQ scores[20].

The significant reduction in serum uric acid levels in the test group is a key finding, as hyperuricemia is the primary pathological driver of gout. This effect can be attributed to the presence of *Morinda citrifolia*, which has been shown to inhibit xanthine oxidase, the enzyme responsible for converting purines to uric acid[21]. Additionally, *Vitex negundo* has been reported to possess uricosuric properties, promoting the excretion of uric acid through the kidneys[22]. These mechanisms likely contributed to the observed reduction in uric acid levels in the test group.

The marked improvements in HRQOL, as measured by the SF-36 questionnaire, in the test group further highlight the clinical benefits of Amrith Noni Artho Plus. The significant improvements in physical functioning, vitality, and mental health domains suggest that the formulation not only alleviates physical symptoms but also enhances overall well-being. This is consistent with previous studies on *Morinda citrifolia*, which has been reported to improve energy levels and reduce fatigue due to its antioxidant properties[23]. The holistic approach of Ayurveda, which addresses both physical and mental health, likely contributed to these improvements.

The safety profile of Amrith Noni Artho Plus was excellent, with no adverse events reported during the study. This is consistent with previous studies on the individual components of the formulation. For instance, *Morinda citrifolia* has been studied for its safety and tolerability, with no significant adverse effects reported in long-term use[24]. Similarly, Shallaki and Guggulu have been shown to be safe and well-tolerated in clinical studies[25,26]. The absence of adverse events in the present study further supports the safety of Amrith Noni Artho Plus for clinical use.

The results of this study suggest that Amrith Noni Artho Plus may offer a safer and more holistic alternative to conventional gout treatments, such as NSAIDs, colchicine, and allopurinol. While these conventional therapies are effective in managing gout symptoms, they are often associated with significant side effects, including gastrointestinal bleeding, renal dysfunction, and hepatotoxicity[27]. In contrast, Amrith Noni Artho Plus demonstrated comparable efficacy in reducing pain and inflammation

without any adverse effects, making it a promising option for long-term gout management.

CONCLUSION

Amrith Noni Artho Plus has proven to be a highly effective and safe option for managing gout flare. The formulation significantly reduces pain, inflammation, and serum uric acid levels while also improving functional ability and overall quality of life. Importantly, no adverse events were reported during the study, and all safety measures remained within normal limits, highlighting its excellent tolerability. Efficacy, safety, and holistic approach of Amrith Noni Artho Plus make it a promising choice for clinical use in managing gout flare, both short-term and long-term.

Declaration by Authors

Ethical Approval: Approved

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REFERENCES

1. Ragab G, Elshahaly M, Bardin T. Gout: An old disease in new perspective - A review. J Adv Res. 2017;8(5):495-511.
2. Dalbeth N, Merriman TR, Stamp LK. Gout. Lancet. 2016; 388(10055):2039-52.
3. Ahmad MI, Masood S, Furlanetto DM, Nicolaou S. Urate Crystals; Beyond Joints. Front Med. 2021; 8:649505.
4. Ahn EY, So MW. The pathogenesis of gout. J Rheum Dis. 2025;32(1):8-16.
5. Gliozzi M, Malara N, Muscoli S, Mollace V. The treatment of hyperuricemia. Int J Cardiol. 2016; 213:23-27.
6. Roddy E, Doherty M. Epidemiology of gout. Arthritis Res Ther. 2010;12(6):223.
7. Tausche AK, Jansen TL, Schröder HE, Bornstein SR, Aringer M, Müller-Ladner U. Gout-current diagnosis and treatment. Dtsch Arztebl Int. 2009;106(34-35):549-555.
8. Punjwani S, Jani C, Liu W, Kakoullis L, Salciccioli I, Al Omari O, Merchant A, Singh H, Marshall D, Shalhoub J, Salciccioli JD, Sehra ST. Burden of gout among different WHO regions, 1990-2019: estimates from the global burden of disease study. Sci Rep. 2024;14(1):15953.
9. GBD 2021 Gout Collaborators. Global, regional, and national burden of gout, 1990-2020, and projections to 2050: a systematic analysis of the Global Burden of Disease Study 2021. Lancet Rheumatol. 2024; 6(8):e507-e517.
10. Roddy E, Bajpai R, Forrester H, Partington RJ, Mallen CD, Clarson LE, Padmanabhan N, Whittle R, Muller S. Safety of colchicine and NSAID prophylaxis when initiating urate-lowering therapy for gout: propensity score-matched cohort studies in the UK Clinical Practice Research Datalink. Ann Rheum Dis. 2023;82(12):1618-1625.
11. Bajpai R, Partington R, Muller S, Forrester H, Mallen CD, Clarson L, Padmanabhan N, Whittle R, Roddy E. Prognostic factors for colchicine prophylaxis-related adverse events when initiating allopurinol for gout: retrospective cohort study. Rheumatology (Oxford). 2025;64(3):1147-1154.
12. Le UQ. The review on medicinal herbs in the treatment of gout through xanthine oxidase inhibitory activity: Call for more research strategy in the future. J Appl Pharm Sci. 2024;14(04):1-13.
13. Lin Z, Gupta JK, Maqbool M, Kumar K, Sharma A, Wahi N. The Therapeutic Management of Chemical and Herbal Medications on Uric Acid Levels and Gout: Modern and Traditional Wisdom. Pharmaceuticals. 2024;17(11):1507.
14. Unno T, Sugimoto A, Kakuda T. Xanthine oxidase inhibitors from the leaves of Lagerstroemia speciosa (L.) Pers. J Ethnopharmacol. 2004;93(2-3):391-395.
15. Dussossoy E, Brat P, Bony E, Boudard F, Poucheret P, Mertz C, Giaimis J, Michel A. Characterization, anti-oxidative and anti-inflammatory effects of Costa Rican noni juice (Morinda citrifolia L.). J Ethnopharmacol. 2011;133(1):108-115.

16. Siddiqui MZ. Boswellia serrata, a potential antiinflammatory agent: an overview. Indian J Pharm Sci. 2011;73(3):255-261.
17. Shin MR, Kim HY, Choi HY, Park KS, Choi HJ, Roh SS. *Boswellia serrata* Extract, 5-Loxin®, Prevents Joint Pain and Cartilage Degeneration in a Rat Model of Osteoarthritis through Inhibition of Inflammatory Responses and Restoration of Matrix Homeostasis. Evid Based Complement Alternat Med. 2022; 2022:3067526.
18. Szapary PO, Wolfe ML, Bloedon LT, Cucchiara AJ, DerMarderosian AH, Cirigliano MD, Rader DJ. Guggulipid for the treatment of hypercholesterolemia: a randomized controlled trial. JAMA. 2003;290(6):765-772.
19. Wang MY, West BJ, Jensen CJ, Nowicki D, Su C, Palu AK, Anderson G. Morinda citrifolia (Noni): a literature review and recent advances in Noni research. Acta Pharmacol Sin. 2002;23(12):1127-1141.
20. Nanda GC, Tiwari RK. Shothahara activities of Dashamoola Dravyas as an anti-inflammatory formulation with special reference to Charak - a review. Ayushdhara. 2016;3(1):479-485.
21. Palu A, Deng S, West B, Jensen J. Xanthine oxidase inhibiting effects of noni (Morinda citrifolia) fruit juice. Phytother Res. 2009;23(12):1790-1791.
22. Dharmasiri MG, Jayakody JR, Galhena G, Liyanage SS, Ratnasooriya WD. Anti-inflammatory and analgesic activities of mature fresh leaves of Vitex negundo. J Ethnopharmacol. 2003;87(2-3):199-206.
23. West BJ, Deng S, Isami F, Uwaya A, Jensen CJ. The Potential Health Benefits of Noni Juice: A Review of Human Intervention Studies. Foods. 2018;7(4):58.
24. Wang MY, Lutfiyya MN, Weidenbacher-Hoper V, Peng L, Lipsky MS, Anderson G. Morinda citrifolia L. (noni) improves the Quality of Life in adults with Osteoarthritis. Funct. Food Health Dis. 2011;1(2):75-90.
25. Singh BB, Mishra LC, Vinjamury SP, Aquilina N, Singh VJ, Shepard N. The effectiveness of Commiphora mukul for osteoarthritis of the knee: an outcomes study. Altern Ther Health Med. 2003;9(3):74-79.
26. Yu G, Xiang W, Zhang T, Zeng L, Yang K, Li J. Effectiveness of Boswellia and Boswellia extract for osteoarthritis patients: a systematic review and meta-analysis. BMC Complement Med Ther. 2020;20(1):225.
27. Drug and Therapeutics Bulletin. Latest guidance on the management of gout. BMJ. 2018;362: k2893.

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