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Comparative Study of Functional Outcomes in Patients with Knee Osteoarthritis Treated with Intra-articular Triamcinolone Acetonide (Kenacort) Alone versus Triamcinolone Acetonide Combined with Hyaluronic Acid

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ABSTRACT

Background: Knee osteoarthritis (OA) is a common degenerative joint disorder associated with chronic pain, functional limitation, and reduced quality of life. Intra-articular corticosteroids provide relatively rapid symptomatic relief, whereas hyaluronic acid (HA) may provide more sustained improvement. Combining these agents may therefore offer additional clinical benefit compared with corticosteroid therapy alone.

Objective: To compare the clinical and functional outcomes of intra-articular triamcinolone acetonide (Kenacort) alone with triamcinolone acetonide combined with hyaluronic acid in patients with symptomatic knee osteoarthritis.

Materials and Methods: This comparative study included 80 patients with Kellgren–Lawrence grade II or III knee OA, divided into two groups of 40 patients each. Group A received intra-articular Kenacort alone, while Group B received Kenacort combined with HA. Pain and functional outcomes were assessed at baseline, 2 weeks, 6 weeks, 3 months, and 6 months using the Visual Analogue Scale (VAS), Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), Knee injury and Osteoarthritis Outcome Score (KOOS), and knee range of motion (ROM). Between-group and within-group changes were evaluated, and longitudinal mixed-effects modelling, multivariable regression, and inverse probability of treatment weighting (IPTW) were used to assess the robustness of treatment effects.

Results: Baseline demographic and clinical characteristics were comparable between the two groups. Both treatments produced significant improvements in VAS and WOMAC scores from baseline ($p<0.001$). However, combination therapy demonstrated greater and more sustained improvement. At 6 months, mean VAS improvement was 2.92±0.87 with Kenacort + HA compared with 1.91±0.81 with Kenacort alone (difference 1.01, 95% CI 0.64–1.39; $p<0.001$). WOMAC improvement was 22.26±6.51 versus 11.90±5.30, respectively (difference 10.36, 95% CI 7.72–13.01; $p<0.001$). Significant treatment-by-time interactions were observed for both VAS ($\beta=-0.275$, $p<0.001$) and WOMAC ($\beta=-2.728$, $p<0.001$), indicating greater sustained improvement with combination therapy. Multivariable and IPTW-adjusted analyses confirmed

significantly better 6-month VAS, WOMAC, and KOOS outcomes with Kenacort + HA (all p<0.001). ROM improvement did not differ significantly between groups (p=0.675). A clinically meaningful combined VAS/WOMAC response was achieved by 85.0% of patients receiving combination therapy compared with 20.0% receiving Kenacort alone (RR=4.25, 95% CI 2.26–8.01; p<0.001; NNT=2). Adverse-event rates were comparable between groups (27.5% vs 22.5%; p=0.797), and no serious adverse events occurred.
Conclusion: Intra-articular Kenacort combined with hyaluronic acid provided significantly greater and more sustained improvement in pain and functional disability than Kenacort alone in patients with grade II–III knee osteoarthritis. The benefit remained significant after multivariable and propensity score-adjusted analyses, while ROM and adverse-event rates were comparable between groups. Combination therapy may therefore provide a more effective option for sustained symptomatic and functional improvement in appropriately selected patients with knee OA.

Keywords*: Knee osteoarthritis; hyaluronic acid; triamcinolone acetonide; Kenacort; intra-articular injection; WOMAC; Visual Analogue Scale; functional outcome.*

INTRODUCTION

Osteoarthritis (OA) is a progressive degenerative joint disorder characterized by deterioration of articular cartilage, alterations in subchondral bone, synovial inflammation, pain, and gradual impairment of joint function [1]. The knee is one of the most frequently affected weight-bearing joints, and knee OA represents an important cause of chronic pain, functional limitation, reduced mobility, and deterioration in quality of life [2]. Its prevalence increases considerably with advancing age, making knee OA a major contributor to musculoskeletal disability worldwide [3–5]. With increasing life expectancy and the growing burden of obesity and other metabolic risk factors, the number of individuals affected by symptomatic knee OA is expected to increase further. Therefore, the principal objectives of treatment are to alleviate pain, improve physical function and mobility, and maintain quality of life while delaying the requirement for surgical intervention [6,7]. Conservative management of knee OA includes lifestyle modification, exercise, weight reduction, physiotherapy, and pharmacological treatment. Commonly prescribed medications include paracetamol, topical or oral non-steroidal anti-inflammatory drugs (NSAIDs), and, in selected patients, opioid analgesics. However, prolonged pharmacological therapy may be limited by adverse effects and poor tolerability. NSAIDs are associated with gastrointestinal, renal, and cardiovascular complications, whereas opioids carry risks of sedation, dependence, and other serious adverse outcomes [8]. Consequently, intra-articular therapies have emerged as important treatment options for patients with persistent symptoms despite conventional conservative management.

Hyaluronic acid (HA) is a naturally occurring high-molecular-weight glycosaminoglycan and an important component of synovial fluid and articular cartilage [7]. It contributes to joint lubrication, shock absorption, maintenance of the viscoelastic properties of synovial fluid, free-radical scavenging, and regulation of several biological processes within the joint [9]. In osteoarthritic joints, progressive depolymerization and reduction in the concentration and molecular weight of endogenous HA compromise the lubricating and viscoelastic properties of synovial fluid [7,9]. Intra-articular administration of HA, commonly referred to as viscosupplementation, is intended to restore these properties and may additionally exert anti-inflammatory and chondroprotective effects. It has consequently been used particularly in patients with mild-to-moderate symptomatic knee OA who remain symptomatic despite initial conservative therapy [10,11].

Intra-articular corticosteroids constitute another commonly used treatment for symptomatic knee OA. Corticosteroids suppress local inflammatory pathways and prostaglandin synthesis and can provide relatively rapid reduction in pain and inflammation [12–15]. Their major advantage is the early onset of symptomatic relief; however, the therapeutic effect may diminish over a comparatively short period [15]. Triamcinolone acetonide (Kenacort) is frequently used as an intra-articular corticosteroid because of its potent anti-inflammatory activity. In contrast, HA may have a slower onset of action but can potentially provide a more sustained symptomatic response. These complementary pharmacological characteristics provide a rationale for combining corticosteroid with HA to obtain both rapid initial pain relief and sustained improvement in joint function.

Evidence regarding combined intra-articular corticosteroid and HA therapy has nevertheless remained variable. Several studies have demonstrated the clinical effectiveness of HA and corticosteroid injections individually, while relatively fewer studies have evaluated their combined administration [16]. In a randomized clinical trial involving 104 patients, de Campos et al. reported that adding triamcinolone to HA resulted in greater early improvement in pain and physical function compared with HA alone, although the differences between the groups diminished during subsequent follow-up [17].

Similarly, Ozturk et al. observed improvement with both HA alone and HA combined with triamcinolone, with the combination producing superior early symptomatic relief [18].
Comparable observations have been reported in other clinical studies. Petrella et al. demonstrated a more rapid reduction in pain following combined HA and corticosteroid administration, although the longer-term outcomes became comparable between treatment groups [19]. Grecomoro et al. likewise observed a faster reduction in pain and earlier improvement in joint mobility when HA was administered with a corticosteroid than when HA was administered alone [20]. An Indian study by Uganath et al. further suggested a synergistic effect of combined treatment, demonstrating significantly greater improvement in pain scores during follow-up in patients receiving both agents [21]. Collectively, these findings suggest that combining HA with a corticosteroid may capitalize on the rapid anti-inflammatory action of corticosteroids while retaining the potentially longer-lasting symptomatic benefits of viscosupplementation.

Despite these encouraging findings, the relative benefit of combined intra-articular therapy remains incompletely established because previous studies have differed in sample size, OA severity, injection protocols, follow-up duration, and outcome assessment [16]. Further comparative evidence is therefore required to determine whether the addition of HA to intra-articular corticosteroid therapy provides a clinically meaningful advantage in terms of pain reduction and functional recovery.

The present study was therefore undertaken to compare the efficacy of intra-articular Kenacort alone with combined intra-articular Kenacort and hyaluronic acid in patients with knee osteoarthritis. The primary objective was to compare improvement in pain and functional outcomes between the two treatment groups during follow-up. It was hypothesized that the addition of hyaluronic acid to Kenacort would provide greater and more sustained symptomatic and functional improvement than Kenacort alone.

MATERIAL AND METHODS

Study Design and Participants

This comparative study included 80 patients with symptomatic primary knee osteoarthritis (OA). Patients were divided into two treatment groups, with 40 patients receiving intra-articular triamcinolone acetonide (Kenacort) alone and 40 patients receiving intra-articular triamcinolone acetonide in combination with hyaluronic acid (HA). Adult patients with clinically and radiologically diagnosed primary knee OA were considered eligible for inclusion. Radiographic severity of OA was assessed using the Kellgren–Lawrence (KL) grading system, and patients with KL grade II or III knee OA were included.

Patients with secondary osteoarthritis, inflammatory arthritis, active local or systemic infection involving the knee, significant associated lower-limb pathology likely to interfere with functional assessment, previous major surgery of the affected knee, known hypersensitivity to the study medications, or any contraindication to intra-articular injection were excluded.

Treatment Groups and Intervention

Patients were categorized into two groups according to the intra-articular treatment received. Group A (n=40) received intra-articular triamcinolone acetonide (Kenacort) alone, whereas Group B (n=40) received intra-articular triamcinolone acetonide combined with hyaluronic acid.

All injections were administered into the affected knee under strict aseptic precautions using a standardized intra-articular technique. The knee was appropriately positioned, the injection site was prepared with an antiseptic solution, and relevant anatomical landmarks were identified before administration of the study medication into the joint space. In patients with clinically significant joint effusion, aspiration was performed before injection when indicated. Following the procedure, patients were observed for immediate complications and were provided standardized post-injection instructions.

Baseline Assessment

A detailed demographic and clinical assessment was performed before administration of the intra-articular injection. Baseline variables included age, sex, body mass index (BMI), duration of osteoarthritis, affected knee, and radiographic KL grade. Baseline pain and functional status were evaluated using the Visual Analogue Scale (VAS), Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), Knee injury and Osteoarthritis Outcome Score (KOOS), and knee range of motion (ROM).

The VAS was used to assess the intensity of knee pain, with higher scores representing greater pain severity. WOMAC was used to assess OA-related pain, stiffness, and functional limitation, with higher scores indicating greater disability. KOOS was expressed on a 0–100 scale, with higher scores representing better knee-related status. Knee ROM was measured in degrees and used as an objective measure of joint mobility.

Outcome Assessment and Follow-up

Clinical outcomes were evaluated at baseline, 2 weeks, 6 weeks, 3 months, and 6 months following intra-articular treatment. VAS, WOMAC, and KOOS scores were recorded at each assessment to evaluate changes in pain, symptoms, and functional status over time. Knee ROM was additionally assessed as an objective functional outcome.
For VAS and WOMAC, improvement was calculated as the baseline score minus the corresponding follow-up score, such that a positive value indicated clinical improvement. As higher KOOS scores represent better clinical status, KOOS improvement was calculated as the follow-up score minus the baseline score. ROM improvement was similarly calculated as the follow-up ROM minus baseline ROM.

The principal outcomes of interest were the changes in VAS, WOMAC, and KOOS during follow-up and the magnitude of improvement at 6 months. ROM was evaluated as an additional objective functional outcome. The trajectory of change across the follow-up period was also compared between the two treatment groups.

Clinically Meaningful Response
A supplementary responder analysis was performed at 6 months. The primary responder definition was based on the prespecified combined criterion of ≥2-point improvement in VAS together with ≥12-point improvement in WOMAC from baseline to 6 months. The proportion of patients satisfying both criteria was calculated for each treatment group.

A supplementary KOOS responder analysis may be reported separately where an appropriate validated or prespecified clinically meaningful improvement threshold for the exact KOOS scoring approach used in the study is available. Such an analysis should be regarded as exploratory if the threshold was not prospectively specified.

Safety Assessment
Treatment-related adverse events were assessed during follow-up. Events specifically recorded included post-injection pain flare, transient knee swelling, local erythema, temporary stiffness, and serious adverse events. The number and proportion of patients experiencing each adverse event and any adverse event were compared between the two treatment groups.

Statistical Analysis
Statistical analysis was performed using appropriate statistical software. Continuous variables were summarized as mean ± standard deviation (SD), while categorical variables were presented as frequency and percentage. Baseline continuous characteristics and outcome measures were compared between treatment groups using Welch's independent-samples t-test, while categorical variables were compared using Fisher's exact test, as appropriate. Within-group changes from baseline to individual follow-up assessments were evaluated using paired-samples t-tests.

For continuous outcomes, between-group differences in improvement were presented as mean differences with 95% confidence intervals (CIs). Hedges' g was additionally calculated to quantify the standardized magnitude of between-group differences, particularly for 6-month changes in VAS, WOMAC, KOOS, and ROM.

To assess longitudinal changes in patient-reported outcomes, patient-level random-intercept linear mixed-effects models were fitted separately for VAS, WOMAC, and KOOS. Treatment group, time, and the treatment × time interaction were included as fixed effects. The treatment × time interaction was used to determine whether the trajectory of clinical improvement differed significantly between the Kenacort-alone and Kenacort + HA groups during follow-up. For VAS and WOMAC, a negative interaction coefficient indicated a greater reduction over time with combination therapy, whereas for KOOS, a positive interaction coefficient indicated greater improvement with combination therapy.

Separate multivariable linear regression models were constructed to evaluate the independent association between treatment group and 6-month VAS, WOMAC, KOOS, and ROM outcomes. Treatment group was entered as the principal independent variable, with adjustment for the corresponding baseline outcome score, age, BMI, and KL grade. Adjusted regression coefficients (β) with 95% CIs and p values were reported.

Propensity Score and IPTW Analysis
As a sensitivity analysis to account for potential baseline differences between treatment groups, propensity score-based inverse probability of treatment weighting (IPTW) was performed. Propensity scores representing the probability of receiving combination therapy were estimated using measured baseline covariates included in the treatment-assignment model. Covariate balance before and after weighting was assessed using standardized mean differences (SMDs). Smaller absolute SMD values after weighting were considered indicative of improved balance between treatment groups.

Following weighting, IPTW-adjusted treatment effects were estimated for the 6-month VAS, WOMAC, KOOS, and ROM outcomes and were expressed as regression coefficients (β) with 95% CIs. These analyses were used to assess whether the observed treatment effects remained consistent after accounting for measured baseline covariate imbalance.

For the clinically meaningful responder outcome, responder proportions were compared between treatment groups, and treatment effects were expressed as relative risks (RRs) with 95% CIs. The number needed to treat (NNT) was calculated from the absolute difference in response rates between the groups.

All statistical tests were two-sided, and a p value <0.05 was considered statistically significant.

Ethical Considerations

The study was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants before enrolment. The study was conducted in accordance with applicable ethical principles for research involving human participants.

RESULTS

Table 1. Baseline Demographic, Clinical, and Disease Characteristics of Patients According to Treatment Group

Characteristic	Kenacort alone (n=40)	Kenacort + HA (n=40)	Total (N=80)	P value
Age, years	58.70 ± 9.83	57.75 ± 7.22	58.23 ± 8.58	0.624
BMI, kg/m²	27.49 ± 3.10	27.97 ± 3.05	27.73 ± 3.06	0.485
OA duration, years	5.05 ± 2.05	4.95 ± 1.61	5.00 ± 1.83	0.809
Baseline VAS	7.23 ± 0.59	7.11 ± 0.76	7.17 ± 0.68	0.430
Baseline WOMAC	60.60 ± 7.18	60.79 ± 8.99	60.70 ± 8.08	0.916
Baseline KOOS	42.66 ± 8.58	44.15 ± 8.03	43.40 ± 8.30	0.424
Baseline ROM, degrees	103.63 ± 8.80	102.54 ± 6.97	103.09 ± 7.91	0.538
Sex				**0.178**
Male	15 (37.5%)	22 (55.0%)	37 (46.2%)	
Female	**25 (62.5%)**	**18 (45.0%)**	**43 (53.8%)**	
Affected knee				**0.823**
Right	22 (55.0%)	20 (50.0%)	42 (52.5%)	
Left	**18 (45.0%)**	**20 (50.0%)**	**38 (47.5%)**	
Kellgren–Lawrence grade				**0.655**
Grade II	**21 (52.5%)**	**18 (45.0%)**	**39 (48.8%)**	
Grade III	19 (47.5%)	22 (55.0%)	41 (51.2%)	

The baseline demographic, clinical, and disease characteristics were generally comparable between the two treatment groups. The mean age was 58.70 ± 9.83 years in the Kenacort-alone group and 57.75 ± 7.22 years in the Kenacort + HA group (p=0.624), while the mean BMI was 27.49 ± 3.10 kg/m² and 27.97 ± 3.05 kg/m², respectively (p=0.485). The mean duration of osteoarthritis was also similar between the groups (5.05 ± 2.05 vs 4.95 ± 1.61 years; p=0.809). No significant baseline differences were observed in VAS (7.23 ± 0.59 vs 7.11 ± 0.76; p=0.430), WOMAC (60.60 ± 7.18 vs 60.79 ± 8.99; p=0.916), KOOS (42.66 ± 8.58 vs 44.15 ± 8.03; p=0.424), or ROM (103.63 ± 8.80° vs 102.54 ± 6.97°; p=0.538). Females constituted 62.5% of the Kenacort-alone group and 45.0% of the Kenacort + HA group, while males constituted 37.5% and 55.0%, respectively; the sex distribution was not statistically different between groups (p=0.178). Similarly, the distribution of the affected knee (right: 55.0% vs 50.0%; left: 45.0% vs 50.0%; p=0.823) and Kellgren–Lawrence grade (grade II: 52.5% vs 45.0%; grade III: 47.5% vs 55.0%; p=0.655) did not differ significantly between the groups. Overall, none of the baseline comparisons reached statistical significance (all p>0.05), although standardized mean differences indicated some pre-weighting covariate imbalance, particularly for sex, which was subsequently addressed using IPTW.

Table 2. Serial Changes in Pain and Functional Outcomes (VAS, WOMAC, and KOOS) During Follow-up According to Treatment Group

Outcome	Time point	Kenacort alone	Kenacort + HA	B−A difference (95% CI)	P value
VAS	Baseline	7.23 ± 0.59	7.11 ± 0.76	-0.12 (-0.42 to 0.18)	0.430
VAS	2 weeks	4.67 ± 0.85	4.36 ± 0.88	-0.31 (-0.69 to 0.08)	0.116
VAS	6 weeks	4.48 ± 0.83	3.96 ± 1.01	-0.53 (-0.94 to -0.11)	0.013
VAS	3 months	5.07 ± 0.89	4.04 ± 1.11	-1.03 (-1.48 to -0.59)	<0.001
VAS	6 months	5.32 ± 0.83	4.19 ± 1.11	-1.13 (-1.57 to -0.70)	<0.001
WOMAC	Baseline	60.60 ± 7.18	60.79 ± 8.99	0.19 (-3.43 to 3.82)	0.916
WOMAC	2 weeks	44.73 ± 8.97	45.10 ± 8.68	0.37 (-3.56 to 4.30)	0.850

Outcome	Time point	Kenacort alone	Kenacort + HA	B−A difference (95% CI)	P value
WOMAC	6 weeks	41.07 ± 7.67	39.57 ± 9.76	-1.50 (-5.41 to 2.41)	0.446
WOMAC	3 months	44.72 ± 9.54	38.53 ± 10.02	-6.19 (-10.54 to -1.83)	0.006
WOMAC	6 months	48.70 ± 7.89	38.53 ± 10.68	-10.17 (-14.36 to -5.98)	<0.001
KOOS	Baseline	42.66 ± 8.58	44.15 ± 8.03	1.49 (-2.21 to 5.19)	0.424
KOOS	2 weeks	56.03 ± 9.61	59.98 ± 9.11	3.95 (-0.21 to 8.12)	0.063
KOOS	6 weeks	60.42 ± 9.77	65.17 ± 9.01	4.75 (0.57 to 8.94)	0.027
KOOS	3 months	57.42 ± 10.49	71.10 ± 8.97	13.68 (9.34 to 18.03)	<0.001
KOOS	6 months	53.56 ± 9.27	71.39 ± 9.57	17.83 (13.63 to 22.02)	<0.001

Both treatment groups demonstrated improvement in pain and functional outcomes during follow-up; however, the magnitude and persistence of improvement increasingly favored the Kenacort + HA group. VAS scores were comparable at 2 weeks (p=0.116), but significantly lower with combination therapy at 6 weeks (3.96 ± 1.01 vs 4.48 ± 0.83; p=0.013), 3 months (4.04 ± 1.11 vs 5.07 ± 0.89; p<0.001), and 6 months (4.19 ± 1.11 vs 5.32 ± 0.83; p<0.001). WOMAC scores showed a similar pattern, with significant between-group differences emerging at 3 months (38.53 ± 10.02 vs 44.72 ± 9.54; p=0.006) and becoming more pronounced at 6 months (38.53 ± 10.68 vs 48.70 ± 7.89; p<0.001). KOOS progressively favored combination therapy, with significant differences at 6 weeks (65.17 ± 9.01 vs 60.42 ± 9.77; p=0.027), 3 months (71.10 ± 8.97 vs 57.42 ± 10.49; p<0.001), and 6 months (71.39 ± 9.57 vs 53.56 ± 9.27; p<0.001). These findings indicate a more sustained improvement in pain and knee-related function with Kenacort + HA.

Table 3. Within-Group Improvement in VAS, WOMAC, and KOOS Scores from Baseline to Each Follow-up Visit

Outcome	Follow-up	Group	Baseline	Follow-up	Mean improvement (95% CI)	P value
VAS	2w	Kenacort alone	7.23 ± 0.59	4.67 ± 0.85	2.56 (2.37 to 2.75)	<0.001
VAS	2w	Kenacort + HA	7.11 ± 0.76	4.36 ± 0.88	2.75 (2.57 to 2.93)	<0.001
VAS	6w	Kenacort alone	7.23 ± 0.59	4.48 ± 0.83	2.75 (2.54 to 2.95)	<0.001
VAS	6w	Kenacort + HA	7.11 ± 0.76	3.96 ± 1.01	3.15 (2.93 to 3.38)	<0.001
VAS	3m	Kenacort alone	7.23 ± 0.59	5.07 ± 0.89	2.16 (1.94 to 2.37)	<0.001
VAS	3m	Kenacort + HA	7.11 ± 0.76	4.04 ± 1.11	3.07 (2.83 to 3.32)	<0.001
VAS	6m	Kenacort alone	7.23 ± 0.59	5.32 ± 0.83	1.91 (1.65 to 2.17)	<0.001
VAS	6m	Kenacort + HA	7.11 ± 0.76	4.19 ± 1.11	2.92 (2.64 to 3.20)	<0.001
WOMAC	2w	Kenacort alone	60.60 ± 7.18	44.73 ± 8.97	15.87 (14.40 to 17.35)	<0.001
WOMAC	2w	Kenacort + HA	60.79 ± 8.99	45.10 ± 8.68	15.69 (14.40 to 16.98)	<0.001
WOMAC	6w	Kenacort alone	60.60 ± 7.18	41.07 ± 7.67	19.53 (17.94 to 21.12)	<0.001
WOMAC	6w	Kenacort + HA	60.79 ± 8.99	39.57 ± 9.76	21.23 (19.63 to 22.82)	<0.001
WOMAC	3m	Kenacort alone	60.60 ± 7.18	44.72 ± 9.54	15.88 (14.26 to 17.50)	<0.001
WOMAC	3m	Kenacort + HA	60.79 ± 8.99	38.53 ± 10.02	22.26 (20.40 to 24.13)	<0.001

Outcome	Follow-up	Group	Baseline	Follow-up	Mean improvement (95% CI)	P value
WOMAC	6m	Kenacort alone	60.60 ± 7.18	48.70 ± 7.89	11.90 (10.20 to 13.59)	<0.001
WOMAC	6m	Kenacort + HA	60.79 ± 8.99	38.53 ± 10.68	22.26 (20.18 to 24.34)	<0.001
KOOS	2w	Kenacort alone	42.66 ± 8.58	56.03 ± 9.61	13.37 (11.94 to 14.81)	<0.001
KOOS	2w	Kenacort + HA	44.15 ± 8.03	59.98 ± 9.11	15.83 (14.45 to 17.21)	<0.001
KOOS	6w	Kenacort alone	42.66 ± 8.58	60.42 ± 9.77	17.76 (16.15 to 19.38)	<0.001
KOOS	6w	Kenacort + HA	44.15 ± 8.03	65.17 ± 9.01	21.02 (19.51 to 22.54)	<0.001
KOOS	3m	Kenacort alone	42.66 ± 8.58	57.42 ± 10.49	14.76 (13.14 to 16.39)	<0.001
KOOS	3m	Kenacort + HA	44.15 ± 8.03	71.10 ± 8.97	26.95 (25.14 to 28.76)	<0.001
KOOS	6m	Kenacort alone	42.66 ± 8.58	53.56 ± 9.27	10.90 (9.23 to 12.57)	<0.001
KOOS	6m	Kenacort + HA	44.15 ± 8.03	71.39 ± 9.57	27.23 (25.29 to 29.18)	<0.001

Significant within-group improvements in VAS, WOMAC, and KOOS were observed at all follow-up assessments in both treatment groups (all p<0.001). In the Kenacort-alone group, VAS improvement reached 2.75 points at 6 weeks but decreased to 1.91 points at 6 months, whereas the Kenacort + HA group maintained a greater 6-month improvement of 2.92 points. Similarly, WOMAC improvement at 6 months was 11.90 points with Kenacort alone compared with 22.26 points with combination therapy. KOOS increased significantly in both groups, but the improvement at 6 months was substantially greater with Kenacort + HA (27.23 points) than with Kenacort alone (10.90 points). Thus, although both interventions produced significant clinical benefit, combination therapy demonstrated greater persistence of pain relief and functional improvement.

Table 4. Between-Group Comparison of 6-Month Improvement in Pain and Functional Outcomes Including KOOS

Outcome	Kenacort alone	Kenacort + HA	Difference in improvement (95% CI)	Hedges g	P value
VAS	1.91 ± 0.81	2.92 ± 0.87	1.01 (0.64 to 1.39)	1.19	<0.001
WOMAC	11.90 ± 5.30	22.26 ± 6.51	10.36 (7.72 to 13.01)	1.73	<0.001
KOOS	10.90 ± 5.23	27.23 ± 6.08	16.33 (13.81 to 18.86)	2.85	<0.001
ROM	8.50 ± 8.19	9.20 ± 6.59	0.70 (-2.61 to 4.01)	0.09	0.675

At 6 months, combination therapy produced significantly greater improvement in pain and patient-reported functional outcomes than Kenacort alone. Mean VAS improvement was 2.92 ± 0.87 with Kenacort + HA compared with 1.91 ± 0.81 with Kenacort alone, yielding a between-group difference of 1.01 points (95% CI 0.64–1.39; p<0.001) and a large effect size (Hedges' g=1.19). WOMAC improvement was also substantially greater with combination therapy (22.26 ± 6.51 vs 11.90 ± 5.30), with a mean difference of 10.36 points (95% CI 7.72–13.01; p<0.001; Hedges' g=1.73). KOOS improvement was 27.23 ± 6.08 versus 10.90 ± 5.23, corresponding to a difference of 16.33 points (95% CI 13.81–18.86; p<0.001) and a very large effect size (Hedges' g=2.85). In contrast, ROM improvement did not differ significantly between groups (9.20 ± 6.59° vs 8.50 ± 8.19°; p=0.675).

Table 5. Longitudinal Mixed-Effects Model for VAS, WOMAC, and KOOS

Outcome	Effect	β	SE	95% CI	P value
VAS	Treatment	-0.074	0.221	-0.508 to 0.360	0.738
VAS	Time	-0.342	0.056	-0.453 to -0.231	<0.001
VAS	Treatment × time	-0.275	0.080	-0.431 to -0.119	<0.001
WOMAC	Treatment	1.998	2.049	-2.017 to 6.014	0.329
WOMAC	Time	-2.381	0.373	-3.113 to -1.649	<0.001
WOMAC	Treatment × time	-2.728	0.528	-3.764 to -1.693	<0.001

Outcome	Effect	β	SE	95% CI	P value
KOOS	Treatment	-0.138	2.071	-4.197 to 3.921	0.947
KOOS	Time	2.320	0.339	1.655 to 2.984	<0.001
KOOS	Treatment × time	4.240	0.479	3.300 to 5.180	<0.001

Longitudinal mixed-effects modelling demonstrated significant changes over time in VAS, WOMAC, and KOOS and showed that the trajectory of improvement differed significantly between the treatment groups. For VAS, the treatment × time interaction was significant (β=-0.275, 95% CI -0.431 to -0.119; p<0.001), indicating a progressively greater reduction in pain with combination therapy. A significant treatment × time interaction was also observed for WOMAC (β=-2.728, 95% CI -3.764 to -1.693; p<0.001), demonstrating greater improvement in functional disability over time in the Kenacort + HA group. For KOOS, the treatment × time coefficient was positive and significant (β=4.240, 95% CI 3.300–5.180; p<0.001), indicating a greater increase in knee-related functional status over time with combination therapy. The non-significant treatment main effects for VAS, WOMAC, and KOOS further indicate that the principal treatment differences developed during follow-up rather than being present at baseline.

Table 6. Multivariable-Adjusted Predictors of 6-Month VAS, WOMAC, KOOS, and ROM Outcomes

Outcome	Predictor	Adjusted β	95% CI	P value
VAS	Kenacort + HA vs alone	-1.040	-1.424 to -0.655	<0.001
VAS	Baseline score	0.777	0.492 to 1.062	<0.001
VAS	Age	-0.013	-0.038 to 0.012	0.297
VAS	BMI	-0.023	-0.094 to 0.047	0.513
VAS	KL grade	-0.184	-0.564 to 0.196	0.338
WOMAC	Kenacort + HA vs alone	-9.997	-12.690 to -7.305	<0.001
WOMAC	Baseline score	0.896	0.731 to 1.062	<0.001
WOMAC	Age	0.131	-0.044 to 0.306	0.139
WOMAC	BMI	0.183	-0.308 to 0.674	0.461
WOMAC	KL grade	0.240	-2.448 to 2.929	0.859
KOOS	Kenacort + HA vs alone	16.472	13.943 to 19.001	<0.001
KOOS	Baseline KOOS	0.888	0.733 to 1.043	<0.001
KOOS	Age	0.046	-0.091 to 0.183	0.502
KOOS	BMI	-0.366	-0.823 to 0.090	0.114
KOOS	KL grade	0.955	-1.599 to 3.510	0.458
ROM	Kenacort + HA vs alone	0.854	-2.640 to 4.348	0.628
ROM	Baseline score	1.010	0.787 to 1.234	<0.001
ROM	Age	0.036	-0.192 to 0.263	0.756
ROM	BMI	-0.039	-0.689 to 0.610	0.904
ROM	KL grade	-0.497	-3.957 to 2.964	0.776

After adjustment for baseline outcome score, age, BMI, and KL grade, treatment with Kenacort + HA remained independently associated with significantly better 6-month pain and functional outcomes. Combination therapy was associated with a 1.04-point lower VAS score than Kenacort alone (adjusted β=-1.040, 95% CI -1.424 to -0.655; p<0.001) and an approximately 10-point lower WOMAC score (β=-9.997, 95% CI -12.690 to -7.305; p<0.001). It was also independently associated with a 16.47-point higher KOOS score (β=16.472, 95% CI 13.943–19.001; p<0.001). Baseline VAS, WOMAC, KOOS, and ROM were significant predictors of their respective 6-month outcomes, whereas age, BMI, and KL grade were not significantly associated with the final outcomes. No independent treatment effect was demonstrated for ROM (β=0.854, 95% CI -2.640 to 4.348; p=0.628).

Table 7. Baseline Covariate Balance Before and After Inverse Probability of Treatment Weighting (IPTW)

Covariate	Unweighted SMD	IPTW SMD
Age	-0.110	-0.002
BMI	0.157	-0.005
OA duration	-0.054	0.004
Baseline VAS	-0.177	0.013
Baseline WOMAC	0.024	-0.000
Baseline KOOS	0.180	0.015
Sex	0.352	0.047
KL grade	0.149	0.026

Baseline covariate balance improved substantially following inverse probability of treatment weighting. Before weighting, the largest imbalance was observed for sex (SMD=0.352), with additional imbalance in baseline KOOS (SMD=0.180), baseline VAS (SMD=-0.177), BMI (SMD=0.157), and KL grade (SMD=0.149). Following IPTW, the absolute SMDs for all included covariates were reduced to below 0.05, including sex (0.047), KL grade (0.026), baseline KOOS (0.015), baseline VAS (0.013), and BMI (-0.005). These findings indicate that IPTW achieved excellent balance in the measured baseline covariates between the two treatment groups.

Table 8. IPTW-Adjusted Effect of Kenacort Plus Hyaluronic Acid versus Kenacort Alone on 6-Month Outcomes

Outcome	Adjusted treatment effect β	95% CI	P value
VAS	-1.037	-1.403 to -0.672	<0.001
WOMAC	-10.202	-12.802 to -7.602	<0.001
KOOS	16.711	12.624 to 20.798	<0.001
ROM	0.829	-2.370 to 4.028	0.612

The IPTW-adjusted analysis confirmed the robustness of the primary findings after accounting for measured baseline differences between the treatment groups. Compared with Kenacort alone, Kenacort + HA was associated with a significantly lower 6-month VAS score (β=-1.037, 95% CI -1.403 to -0.672; p<0.001) and WOMAC score (β=-10.202, 95% CI -12.802 to -7.602; p<0.001). Combination therapy was additionally associated with a 16.71-point higher KOOS score (β=16.711, 95% CI 12.624–20.798; p<0.001). In contrast, the IPTW-adjusted difference in ROM remained non-significant (β=0.829, 95% CI -2.370 to 4.028; p=0.612). The consistency between conventional multivariable and propensity-weighted analyses supports a treatment advantage for combination therapy in pain and patient-reported functional outcomes.

Table 9. Clinically Meaningful Treatment Response at 6 Months

Measure	Kenacort alone	Kenacort + HA	Effect estimate	P value
Existing VAS + WOMAC responder	8/40 (20.0%)	34/40 (85.0%)	RR 4.25 (2.26–8.01); NNT 2	<0.001

A significantly greater proportion of patients receiving Kenacort + HA achieved the predefined combined VAS and WOMAC response at 6 months. A clinically meaningful response was observed in 34/40 (85.0%) patients in the combination group compared with 8/40 (20.0%) in the Kenacort-alone group, corresponding to a relative risk of 4.25 (95% CI 2.26–8.01; p<0.001) and an NNT of 2.

Table 10. Frequency and Comparison of Treatment-Related Adverse Events According to Treatment Group

Adverse event	Kenacort alone	Kenacort + HA	P value
Post-injection pain flare	5 (12.5%)	6 (15.0%)	1.000
Transient swelling	3 (7.5%)	5 (12.5%)	0.712
Local erythema	2 (5.0%)	1 (2.5%)	1.000
Temporary stiffness	2 (5.0%)	2 (5.0%)	1.000
Any adverse event	9 (22.5%)	11 (27.5%)	0.797
Serious adverse event	0 (0.0%)	0 (0.0%)	1.000

Both treatment strategies were generally well tolerated, with adverse events being infrequent, mild, and comparable between groups. At least one adverse event was reported in 9/40 (22.5%) patients receiving Kenacort alone and 11/40 (27.5%) receiving Kenacort + HA (p=0.797). Post-injection pain flare occurred in 12.5% versus 15.0%, transient swelling in 7.5% versus 12.5%, local erythema in 5.0% versus 2.5%, and temporary stiffness in 5.0% of patients in each group, respectively; none of these differences was statistically significant. Importantly, no serious adverse events were reported in either treatment group, suggesting a comparable short-term safety profile for the two interventions.

DISCUSSION

The present study compared intra-articular triamcinolone acetonide (Kenacort) alone with triamcinolone combined with hyaluronic acid (HA) in patients with symptomatic knee osteoarthritis and demonstrated that both treatment strategies produced significant improvement, but combination therapy provided greater and more sustained benefit in pain and functional outcomes. No statistically significant differences in baseline demographic, clinical, or disease characteristics were observed between the treatment groups. However, standardized mean differences indicated some pre-weighting covariate imbalance, particularly for sex. IPTW substantially improved covariate balance, with absolute SMDs below 0.05 for all included variables.

Both groups showed marked early reduction in pain. At 2 weeks, VAS decreased substantially in both groups, with no statistically significant between-group difference. This is consistent with the known rapid anti-inflammatory action of intra-articular corticosteroids. Arroll and Goodyear-Smith [22], Bellamy et al. [23], Godwin and Dawes [24], and Jüni et al. [25] have shown that corticosteroid injections provide clinically useful short-term pain relief in knee OA, although the effect

generally diminishes over time. Richards et al. [26] and Nguyen et al. [27] also emphasized that corticosteroids are particularly useful when inflammatory symptoms such as synovitis, swelling, and acute pain predominate.

The major difference between the two treatment groups emerged with longer follow-up. In the present study, VAS became significantly lower in the Kenacort + HA group by 6 weeks and remained significantly lower at 3 and 6 months. At 6 months, mean VAS improvement was 2.92 points with combination therapy compared with 1.91 points with Kenacort alone. These findings are in agreement with the therapeutic trajectory described by Bannuru et al. [28], who demonstrated that corticosteroids tend to have greater early efficacy whereas HA becomes relatively more effective after several weeks. Askari et al. [29] similarly reported that both HA and corticosteroids reduce pain initially, but the effect of HA is more durable. Leighton et al. [30] also found longer-lasting efficacy of HA compared with methylprednisolone, while Caborn et al. [31] reported sustained benefit with hylan G-F 20 compared with triamcinolone. Thus, the later separation seen in our study is biologically and clinically plausible because both groups received corticosteroid, but only the combination group additionally received the longer-duration viscosupplementation effect of HA.

Functional outcomes showed a similar pattern. WOMAC improved significantly in both groups, but the difference between groups became significant at 3 months and was most pronounced at 6 months. Mean WOMAC improvement was 22.26 points with Kenacort + HA compared with 11.90 points with Kenacort alone. McConnell et al. [32] established WOMAC as a reliable measure of pain, stiffness, and physical function in OA, and Askari et al. [29] also used WOMAC to compare intra-articular HA and corticosteroid therapy. Their study demonstrated functional improvement after both interventions, with HA providing a more sustained response. Vincent et al. [33] further reported that HA viscosupplementation may influence synovial inflammation, which may partly explain prolonged improvement in symptoms and physical function.
KOOS findings strengthened these observations. Baseline KOOS was comparable between groups, but significantly higher scores were seen in the combination group from 6 weeks onward. At 6 months, KOOS improvement was 27.23 points with Kenacort + HA compared with 10.90 points with Kenacort alone. Roos and Lohmander [34] developed KOOS as a comprehensive knee-specific patient-reported outcome measure, while Peer and Lane [35] and van Meer et al. [36] demonstrated its value in assessing symptoms, activities, and functional recovery. Askari et al. [29] also reported that KOOS symptoms and activities of daily living improved following both HA and corticosteroid injections. In our study, the much greater improvement in KOOS with combination therapy indicates that the treatment advantage extended beyond pain reduction to broader knee-related function and patient-perceived status.

The consistency of VAS, WOMAC, and KOOS findings is important. All three outcomes showed a similar trajectory: strong early improvement in both groups, followed by partial loss of benefit with Kenacort alone and greater persistence of improvement with Kenacort + HA. This pattern was confirmed by mixed-effects modelling, in which treatment-by-time interactions were significant for VAS, WOMAC, and KOOS. The non-significant treatment main effects indicate that the groups were similar at baseline and that the treatment advantage developed over time.

The mechanism underlying this response is likely related to the complementary actions of corticosteroid and HA. Corticosteroids rapidly suppress synovial inflammation, inflammatory-cell migration, edema, and inflammatory mediator production. Robinson et al. [37] highlighted low-grade inflammation as an important contributor to OA pathogenesis, while Vandeweerd et al. [38] and Tehranzadeh et al. [39] described the effects of corticosteroids on cartilage metabolism and inflammatory pathways. However, repeated or high-dose corticosteroid exposure may adversely influence proteoglycan and HA synthesis, and its symptomatic effect tends to be relatively short-lived. In contrast, HA improves the viscoelastic properties of synovial fluid, contributes to lubrication and shock absorption, and may exert anti-inflammatory, analgesic, and chondroprotective effects. Bellamy et al. [40], Strand et al. [41], Colen et al. [42], Ray [43], McArthur et al. [44], and Henrotin et al. [45] have all reported sustained symptomatic benefits with viscosupplementation in appropriately selected patients.

Studies evaluating combined HA and corticosteroid therapy have also shown complementary effects. Previous trials reported more rapid pain relief when corticosteroid was added to HA, although in some studies the difference diminished by 6 months. This differs from the present study because our comparator was corticosteroid alone rather than HA alone. Therefore, both groups received the early anti-inflammatory effect, while only the combination group received the additional sustained HA effect. This difference in study design explains why the treatment advantage in our study became more apparent at later follow-up.

At 6 months, the magnitude of treatment benefit was substantial. Hedges' g values were 1.19 for VAS, 1.73 for WOMAC, and 2.85 for KOOS, indicating large to very large standardized treatment effects. Multivariable regression also confirmed that Kenacort + HA remained independently associated with lower VAS, lower WOMAC, and higher KOOS after adjustment for baseline score, age, BMI, and KL grade. Importantly, IPTW analysis produced very similar treatment effects after achieving excellent balance in measured baseline covariates. The agreement between unadjusted comparisons, mixed-effects models, multivariable regression, and propensity-weighted analysis strengthens the consistency of the findings.

Responder analysis also suggested a clinically important advantage. At 6 months, 85% of patients receiving combination therapy achieved the predefined VAS + WOMAC response compared with 20% receiving Kenacort alone, corresponding to an RR of 4.25 and an NNT of 2.

In contrast to the patient-reported outcomes, ROM improvement did not differ significantly between groups. Both groups improved, but the mean difference at 6 months was small and non-significant. This is consistent with previous observations that pain and functional perception may improve more substantially than objective range of motion after intra-articular therapy. Skwara et al. [46] demonstrated that clinical and gait outcomes do not always change proportionately, while studies of HA and corticosteroid combinations have also reported improvement in flexion without persistent significant between-group differences. Structural abnormalities such as osteophytes, capsular stiffness, deformity, and chronic contracture may limit ROM despite improved pain control.

Safety outcomes were comparable. Mild adverse events such as transient pain flare, swelling, erythema, and temporary stiffness occurred at similar frequencies in both groups, and no serious adverse events were observed. These findings are consistent with Strand et al. [41], McArthur et al. [44], Adams et al. [47], and Maheu et al. [48], who reported acceptable safety profiles for HA injections, and with the broader corticosteroid literature showing predominantly transient local adverse effects. Nevertheless, repeated corticosteroid exposure should be used judiciously because of potential concerns regarding cartilage metabolism and infection risk before subsequent arthroplasty, as discussed by Vandeweerd et al. [38], Marsland et al. [49], and Papavasiliou et al. [50].

Overall, the present study supports the concept that intra-articular triamcinolone provides rapid symptomatic benefit, while the addition of HA appears to prolong and amplify improvement in pain and functional status. The findings are consistent with previous studies by Bannuru et al. [28], Askari et al. [29], Leighton et al. [30], Caborn et al. [31], Bellamy et al. [40], Strand et al. [41], and others demonstrating that HA has a more sustained therapeutic trajectory than corticosteroid alone. The main strength of the present study is the consistent advantage observed across VAS, WOMAC, and KOOS, supported by repeated-measures modelling, multivariable adjustment, IPTW analysis, and responder outcomes. Larger randomized multicenter studies with longer follow-up, standardized HA formulations, individual KOOS subscales, and structural imaging or cartilage biomarkers are required to confirm the durability and long-term clinical significance of these findings.

CONCLUSION

The present study demonstrates that both intra-articular triamcinolone acetonide (Kenacort) alone and Kenacort combined with hyaluronic acid are effective in reducing pain and improving functional outcomes in patients with Kellgren–Lawrence grade II–III knee osteoarthritis. However, the combination of Kenacort with hyaluronic acid was associated with significantly greater and more sustained improvement in VAS, WOMAC, and KOOS outcomes, particularly at 3 and 6 months. These findings remained consistent after multivariable adjustment and propensity score-based IPTW analysis, supporting the robustness of the observed treatment effect. Although improvement in knee range of motion was comparable between the groups, a substantially greater proportion of patients receiving combination therapy achieved a clinically meaningful improvement in pain and function. Both treatment approaches were well tolerated, with comparable rates of minor adverse events and no serious adverse events. Thus, intra-articular Kenacort combined with hyaluronic acid was associated with greater sustained pain relief and improvement in patient-reported functional outcomes than Kenacort alone in patients with symptomatic grade II–III knee osteoarthritis.

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