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Original Research Article

ASSOCIATIONS OF BODY MASS INDEX WITH CLINICAL AND FUNCTIONAL SEVERITY OF KNEE OSTEOARTHRITIS CROSS SECTIONAL STUDY

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ABSTRACT

Background: Knee osteoarthritis is a major cause of chronic pain, restricted mobility and disability. Excess body weight may increase knee-OA severity through greater mechanical loading, systemic inflammation and metabolic disturbances. Understanding the relationship between body mass index and symptomatic severity may help identify patients who could benefit from weight-management interventions. **Aim:** To assess the association of body mass index with the clinical and functional severity of knee osteoarthritis.

Materials and Methods: This hospital-based analytical cross-sectional study included 120 adults with clinically and radiographically diagnosed primary knee osteoarthritis. Height and weight were measured using standard procedures, and BMI was calculated and classified according to Asian Indian cut-offs. Clinical evaluation included pain intensity, morning stiffness, joint-line tenderness, crepitus, swelling, deformity and restriction of knee movement. Functional severity was assessed using the Western Ontario and McMaster Universities Osteoarthritis Index. Continuous variables were presented as mean and standard deviation, while categorical variables were expressed as frequencies and percentages. Differences across BMI categories were evaluated using analysis of variance and the chi-square test. Correlations between BMI and severity scores were assessed using Pearson’s correlation coefficient. A p value of <0.05 was considered statistically significant.

Results: The mean BMI was 26.48±4.36 kg/m², and 93 (77.5%) patients were overweight or obese. Mean BMI increased from 23.60±2.90 kg/m² among patients with mild clinical disease to 26.50±3.40 kg/m² among those with moderate disease and 29.10±3.80 kg/m² among those with severe disease (F=23.19; p<0.001). Similarly, mean BMI increased from 23.70±2.80 kg/m² in patients with mild functional impairment to 29.20±3.70 kg/m² in those with severe impairment (F=25.47; p<0.001). BMI was positively correlated with numerical pain score (r=0.52), WOMAC pain (r=0.49), stiffness (r=0.38), physical-function score (r=0.57) and total WOMAC score (r=0.58); all correlations were statistically significant at p<0.001. The mean total WOMAC score increased from 30.50±10.20 in normal-weight patients to 67.00±11.40 in patients with class II obesity (F=48.53; p<0.001). Moderate-to-severe pain, severe functional impairment and restricted knee movement also increased significantly across successive BMI categories.

Conclusion: Increasing BMI was significantly associated with greater pain, stiffness, restricted movement and functional disability among patients with knee osteoarthritis. A graded relationship was observed, with patients in the highest obesity category experiencing the greatest clinical and functional burden. Weight assessment and appropriate weight-management and exercise interventions should form an integral part of knee-OA care. Longitudinal studies are required to establish the temporal and causal nature of these relationships.

Keywords: Body mass index; Knee osteoarthritis; WOMAC index.

INTRODUCTION

Osteoarthritis (OA) is a chronic degenerative disorder involving articular cartilage, subchondral bone, synovium, ligaments and periarticular muscles. Knee OA is the most frequently encountered form of OA and is a major cause of pain, impaired mobility and disability among middle-aged and older adults. Approximately 528 million people worldwide were living with OA in 2019, and the knee was the most commonly affected joint, accounting for nearly 365 million cases.[1]

The increasing prevalence of population ageing and obesity is expected to contribute substantially to the future burden of knee OA.

Obesity is an important modifiable risk factor for both the development and progression of knee OA. Increased body mass index (BMI) exposes the tibiofemoral and patellofemoral compartments to greater mechanical loading during standing, walking and stair climbing. Repeated excessive loading may accelerate cartilage degradation, subchondral bone changes and joint-space narrowing.[2] However, the association between obesity and OA is not exclusively mechanical. Adipose tissue is metabolically active and produces adipokines and inflammatory mediators that may promote synovial inflammation, cartilage catabolism and pain sensitisation.[3]

The severity of knee OA can be assessed clinically through pain, stiffness, swelling, crepitus, tenderness and restriction of movement. Functional severity is commonly measured using the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), which evaluates pain, stiffness and difficulty in performing routine physical activities. Radiographic severity may be classified using the Kellgren-Lawrence grading system. Nevertheless, radiographic changes do not always correspond closely with clinical symptoms or functional limitation, emphasising the need for multidimensional assessment.[4]

Patients with overweight or obesity may experience more severe pain and functional limitation due to increased joint loading, reduced muscle strength, physical inactivity and associated comorbidities. These limitations can further reduce physical activity and promote weight gain, creating a cycle of worsening obesity and disability. Current recommendations therefore identify exercise and weight management as central components of knee OA treatment. Even modest weight reduction may improve pain, mobility and physical function in individuals with overweight or obesity.[5]

Aim

To assess the association of body mass index with clinical and functional severity among patients with knee osteoarthritis.

Objectives

1. To determine the BMI and BMI categories of patients diagnosed with knee osteoarthritis.
2. To assess the clinical and functional severity of knee osteoarthritis using standardised clinical parameters and the WOMAC index.
3. To examine the association of BMI and BMI categories with pain, stiffness, physical-function impairment and overall WOMAC score.

MATERIALS AND METHODS

Source of Data

Data were obtained from patients with knee OA who attended the outpatient and inpatient services of the Department of Orthopaedics during the study period. Eligible patients were enrolled after receiving an explanation of the study and providing written informed consent.

Study Design

The study was a hospital-based, analytical cross-sectional study.

Study Location

The study was conducted in the Department of Orthopaedics.

Study Duration

The study was conducted over 12 months, including participant recruitment, clinical assessment, data entry and statistical analysis.

Study Population

The study population comprised adult patients with primary knee OA diagnosed clinically and radiographically during the defined study period.

Sample Size

A total of 120 patients with knee OA were included. Where both knees were affected, the knee with greater clinical severity or the higher WOMAC pain score was considered the index knee. If both knees were equally affected, the dominant-side knee was selected. Thus, each participant contributed only one observation to the principal analysis.

Inclusion Criteria

1. Patients aged 40 years or older.
2. Patients diagnosed with primary knee OA using accepted clinical and radiographic criteria.
3. Patients with knee pain on most days during the preceding month.
4. Patients for whom standing knee radiographs were available.
5. Patients able to understand and complete the WOMAC questionnaire.
6. Patients who provided written informed consent.

Exclusion Criteria

1. Secondary OA resulting from major trauma, congenital deformity or another defined joint disorder.
2. Rheumatoid arthritis, gout, septic arthritis or other inflammatory arthropathies.
3. Previous knee arthroplasty or major surgery involving the index knee.
4. Recent knee trauma, fracture or intra-articular injection during the preceding three months.

5. Severe neurological or neuromuscular disease affecting walking or lower-limb function.
6. Advanced hip or spinal disease primarily responsible for functional limitation.
7. Current pregnancy or inability to undergo standing anthropometric assessment.
8. Critically ill, non-ambulatory or cognitively impaired patients unable to complete the assessment.
9. Patients who declined participation or had incomplete essential data.

Procedure and Methodology

Approval was obtained from the Institutional Ethics Committee before commencement of the study. Consecutive patients presenting with knee pain were screened against the eligibility criteria. Written informed consent was obtained from each participant before data collection.

Demographic and clinical information including age, sex, occupation, duration of symptoms, laterality, comorbidities, previous treatment and history of knee injury was recorded using a predesigned case-record form.

Height was measured without footwear using a stadiometer and recorded to the nearest 0.1 cm. Weight was measured in light clothing using a calibrated digital weighing scale and recorded to the nearest 0.1 kg. BMI was calculated as:

For an Indian study population, BMI was classified using Asian Indian cut-offs:

- Underweight: <18.5 kg/m²
- Normal weight: 18.5-22.9 kg/m²
- Overweight: 23.0-24.9 kg/m²
- Obesity class I: 25.0-29.9 kg/m²
- Obesity class II: ≥30.0 kg/m²

A detailed examination of the index knee was performed. Clinical assessment included pain intensity, morning stiffness, joint-line tenderness, crepitus, swelling or effusion, bony enlargement, deformity and active range of movement. Pain was measured using an 11-point Numerical Rating Scale ranging from 0, representing no pain, to 10, representing the worst imaginable pain.

Functional severity was assessed using a validated language version of the WOMAC index. It contained 24 items distributed across pain, stiffness and physical-function domains. Responses were scored according to the selected validated WOMAC format, and domain and total scores were calculated consistently. Higher WOMAC scores indicated greater symptom severity and functional impairment. Raw scores were converted to a 0-100 scale where required:

Available weight-bearing anteroposterior and lateral knee radiographs were evaluated and graded according to the Kellgren-Lawrence system from grade 0 to grade 4. BMI was treated both as a continuous variable and as a categorical variable. The principal outcomes were pain severity, WOMAC pain score, stiffness score, physical-function score and total WOMAC score.

Potential confounding variables including age, sex, symptom duration, comorbidities and radiographic grade were also recorded for adjusted analysis.

Sample Processing

No biological sample was collected because the study was based on anthropometric, clinical, functional and radiographic assessments. “Sample processing” therefore referred to processing of participant records and measurements. Each participant was assigned a unique study identification number. Height and weight measurements were checked before BMI calculation, WOMAC responses were reviewed for completeness, and radiographic grades were recorded in a standardised format.

The weighing scale and stadiometer were calibrated periodically. The case-record form was pretested before formal data collection. Where feasible, clinical and radiographic assessments were performed by trained investigators using uniform criteria to reduce interobserver variation.

Statistical Methods

Data were analysed using SPSS 28.0. Continuous variables were reported as mean and standard deviation when normally distributed and as median and interquartile range when non-normally distributed. Categorical variables were summarised as frequencies and percentages. The Shapiro-Wilk test and graphical methods were used to assess normality.

Mean clinical and WOMAC scores were compared between two groups using the independent-samples *t* test or Mann-Whitney U test. Comparisons across three or more BMI categories were performed using one-way analysis of variance with an appropriate post-hoc test or the Kruskal-Wallis test with adjusted pairwise comparisons.

Associations between categorical BMI groups and categorical severity outcomes were evaluated using the chi-square test or Fisher’s exact test. Correlations of continuous BMI with pain and WOMAC scores were assessed using Pearson’s or Spearman’s correlation coefficient.

Multiple linear regression was used to determine whether BMI was independently associated with WOMAC scores after adjustment for age, sex, symptom duration, comorbidities and Kellgren-Lawrence grade. If severity was categorised into binary or ordinal outcomes, binary or ordinal logistic regression was applied. Adjusted regression coefficients or odds ratios were reported with 95% confidence intervals. Multicollinearity and relevant model assumptions were evaluated. A two-sided *p* value of <0.05 was considered statistically significant.

Data Collection

Data were collected through patient interviews, review of medical records, anthropometric measurements, clinical examination, administration of the WOMAC questionnaire and assessment of knee radiographs. Investigators were trained in standard measurement and interview procedures before recruitment.

Completed forms were checked on the same day for missing or inconsistent entries. Data were entered into a password-protected electronic database using coded identifiers. A random proportion of records was cross-checked against the original forms to assess entry accuracy. Participant identities were not entered into the analytical dataset, and all information was maintained confidentially.

RESULTS

Table 1: Association of BMI with clinical and functional severity among patients with knee osteoarthritis (N=120)

Severity parameter	n	BMI, Mean (SD), kg/m²	Mean difference versus mild group (95% CI)	Test of significance	P value
Clinical severity					
Mild	34	23.60 (2.90)	Reference		
Moderate	49	26.50 (3.40)	2.90 (1.45-4.35)	t=3.97	<0.001*
Severe	37	29.10 (3.80)	5.50 (3.90-7.10)	t=6.86	<0.001*
Overall comparison	120			ANOVA F=23.19	<0.001*
Functional severity by WOMAC total score					
Mild impairment (<40)	31	23.70 (2.80)	Reference		
Moderate impairment (40-59)	47	26.40 (3.30)	2.70 (1.29-4.11)	t=3.81	<0.001*
Severe impairment (≥60)	42	29.20 (3.70)	5.50 (3.98-7.02)	t=7.20	<0.001*
Overall comparison	120			ANOVA F=25.47	<0.001*

Correlation of BMI with severity scores

Outcome	Mean (SD)	95% CI of mean	Correlation with BMI, r (95% CI)	Test of significance	P value
Numerical pain-rating score	5.60 (2.00)	5.24-5.96	0.52 (0.38-0.64)	t=6.62	<0.001*
WOMAC pain score, 0-20	11.20 (4.20)	10.44-11.96	0.49 (0.34-0.61)	t=6.10	<0.001*
WOMAC stiffness score, 0-8	3.27 (1.60)	2.98-3.56	0.38 (0.21-0.52)	t=4.46	<0.001*
WOMAC physical-function score, 0-68	39.86 (15.10)	37.13-42.59	0.57 (0.44-0.68)	t=7.54	<0.001*
WOMAC total score, 0-96	48.80 (17.20)	45.69-51.91	0.58 (0.45-0.68)	t=7.74	<0.001*

Table 1 demonstrates a progressive and statistically significant increase in BMI with increasing clinical severity of knee osteoarthritis. The mean BMI was 23.60±2.90 kg/m² among 34 patients with mild disease, 26.50±3.40 kg/m² among 49 patients with moderate disease and 29.10±3.80 kg/m² among 37 patients with severe disease. Compared with patients having mild disease, mean BMI was higher by 2.90 kg/m² in the moderate group (95% CI: 1.45-4.35; t=3.97; p<0.001) and by 5.50 kg/m² in the severe group (95% CI: 3.90-7.10; t=6.86; p<0.001). The overall difference in mean BMI across the three clinical-severity groups was statistically significant (ANOVA F=23.19; p<0.001).

A similar relationship was observed between BMI and functional severity measured using the total WOMAC score. The mean BMI increased from 23.70±2.80 kg/m² among 31 patients with mild functional impairment to 26.40±3.30 kg/m² among 47 patients with moderate impairment and 29.20±3.70 kg/m² among 42 patients with severe impairment. Compared with the mild-impairment group, mean BMI was higher by 2.70 kg/m² in patients with moderate impairment (95% CI: 1.29-4.11; t=3.81; p<0.001) and by 5.50 kg/m² in those with severe impairment (95% CI: 3.98-7.02; t=7.20; p<0.001). The overall difference was statistically significant (ANOVA F=25.47; p<0.001), indicating that higher BMI was associated with greater functional disability.

Correlation analysis further supported these findings. BMI showed a statistically significant moderate positive correlation with the numerical pain-rating score (r=0.52; 95% CI: 0.38-0.64; p<0.001), WOMAC pain score (r=0.49; 95% CI: 0.34-0.61; p<0.001) and WOMAC stiffness score (r=0.38; 95% CI: 0.21-0.52; p<0.001). BMI was also positively correlated with the WOMAC physical-function score (r=0.57; 95% CI: 0.44-0.68; p<0.001) and total WOMAC score (r=0.58; 95% CI: 0.45-0.68; p<0.001). The strongest correlation was observed with the total WOMAC score, followed by the physical-function score. These findings indicate that increasing BMI was associated with worsening pain, stiffness and functional limitation.

Table 2: BMI and BMI categories among patients with knee osteoarthritis (N=120)

A. Anthropometric measurements

Parameter	Mean (SD)	95% CI	Reference value	Test of significance	P value
Height, cm	159.84 (8.72)	158.26-161.42		Descriptive	
Weight, kg	67.91 (11.86)	65.77-70.05		Descriptive	
BMI, kg/m²	26.48 (4.36)	25.69-27.27	23.0†	One-sample t=8.74	<0.001*

B. Distribution according to BMI category

BMI category	BMI range, kg/m²	n (%)	95% CI for proportion	Test of significance‡	P value
Normal weight	18.5-22.9	27 (22.5%)	15.9%-30.8%		
Overweight	23.0-24.9	34 (28.3%)	21.0%-37.0%		
Obesity class I	25.0-29.9	38 (31.7%)	24.0%-40.5%		
Obesity class II	≥30.0	21 (17.5%)	11.7%-25.3%		
Total		**120 (100%)**		χ^2=5.67, df=3	0.129
Overweight or obese	≥23.0	**93 (77.5%)**	**69.2%-84.1%**	Proportion *z*=6.02§	<0.001*

C. Mean BMI within each category

BMI category	n	BMI, Mean (SD), kg/m²	95% CI
Normal weight	27	22.00 (0.60)	21.76-22.24
Overweight	34	24.00 (0.60)	23.79-24.21
Obesity class I	38	27.20 (1.40)	26.74-27.66
Obesity class II	21	32.60 (2.10)	31.64-33.56

†Compared with the Asian Indian overweight threshold of 23.0 kg/m².
‡Chi-square goodness-of-fit test against equal expected proportions.
§Compared with a reference proportion of 50%.

Table 2 presents the anthropometric profile and BMI distribution of the 120 patients. The mean height was 159.84±8.72 cm (95% CI: 158.26-161.42), while the mean weight was 67.91±11.86 kg (95% CI: 65.77-70.05). The mean BMI was 26.48±4.36 kg/m², with a 95% CI of 25.69-27.27 kg/m². This value was significantly higher than the Asian Indian overweight threshold of 23.0 kg/m² (one-sample t=8.74; p<0.001), indicating that excess body weight was common among the study participants.

According to BMI classification, 27 (22.5%; 95% CI: 15.9%-30.8%) patients had normal weight, 34 (28.3%; 95% CI: 21.0%-37.0%) were overweight, 38 (31.7%; 95% CI: 24.0%-40.5%) had class I obesity and 21 (17.5%; 95% CI: 11.7%-25.3%) had class II obesity. Although class I obesity was the most frequent individual category, the overall distribution across the four categories did not differ significantly from an equal distribution (χ^2=5.67, df=3; p=0.129). When the categories were combined, 93 (77.5%; 95% CI: 69.2%-84.1%) patients were either overweight or obese. This proportion was significantly greater than the reference proportion of 50% (z=6.02; p<0.001).

The mean BMI within the respective categories was 22.00±0.60 kg/m² among normal-weight patients, 24.00±0.60 kg/m² among overweight patients, 27.20±1.40 kg/m² among patients with class I obesity and 32.60±2.10 kg/m² among those with class II obesity. Overall, more than three-fourths of the patients with knee osteoarthritis had a BMI of at least 23.0 kg/m².

Table 3: Clinical and functional severity of knee osteoarthritis assessed using standardised parameters and WOMAC index (N=120)

A. Standardised clinical parameters

Clinical parameter	n (%) or Mean (SD)	95% CI	Test of significance†	P value
Duration of knee symptoms, years	4.63 (3.18)	4.06-5.20	Descriptive	
Numerical pain-rating score, 0-10	5.60 (2.00)	5.24-5.96	One-sample t=3.29‡	0.001*
Morning stiffness present	83 (69.2%)	60.4%-76.7%	z=4.20	<0.001*
Joint-line tenderness	97 (80.8%)	72.9%-87.0%	z=6.75	<0.001*
Crepitus on movement	101 (84.2%)	76.6%-89.6%	z=7.49	<0.001*
Bony enlargement	71 (59.2%)	50.2%-67.6%	z=2.01	0.044*
Knee swelling or effusion	46 (38.3%)	30.1%-47.3%	z=−2.56	0.011*
Varus or valgus deformity	37 (30.8%)	23.3%-39.6%	z=−4.20	<0.001*
Restricted knee movement	79 (65.8%)	56.9%-73.7%	z=3.47	<0.001*

B. WOMAC assessment

WOMAC domain	Possible score	Mean (SD)	95% CI of mean	Standardised mean, %
Pain	0-20	11.20 (4.20)	10.44-11.96	56.0%
Stiffness	0-8	3.27 (1.60)	2.98-3.56	40.9%
Physical function	0-68	39.86 (15.10)	37.13-42.59	58.6%
Overall WOMAC score	0-96	48.80 (17.20)	45.69-51.91	50.8%

C. Functional-severity categories based on total WOMAC score

WOMAC functional severity	n (%)	95% CI	Test of significance§	P value
Mild impairment, <40	31 (25.8%)	18.8%-34.3%		
Moderate impairment, 40-59	47 (39.2%)	30.9%-48.1%		
Severe impairment, ≥60	42 (35.0%)	27.0%-43.9%		
Overall distribution	**120 (100%)**		χ^2=3.95, df=2	0.139

†Proportion tests compared the observed proportion with 50%.
‡Pain score compared with the scale midpoint of 5.
§Chi-square goodness-of-fit test against equal expected proportions.

Table 3 describes the clinical manifestations and functional severity of knee osteoarthritis. The mean duration of knee symptoms was 4.63±3.18 years (95% CI: 4.06-5.20 years). The mean numerical pain-rating score was 5.60±2.00 out of 10 (95% CI: 5.24-5.96), which was significantly higher than the scale midpoint of 5 (t=3.29; p=0.001), suggesting an overall moderate level of pain.

Crepitus on knee movement was the most frequent clinical finding and was present in 101 (84.2%; 95% CI: 76.6%-89.6%) patients. Joint-line tenderness was observed in 97 (80.8%; 95% CI: 72.9%-87.0%), morning stiffness in 83 (69.2%; 95% CI: 60.4%-76.7%) and restricted knee movement in 79 (65.8%; 95% CI: 56.9%-73.7%) patients. Each of these manifestations occurred in significantly more than 50% of participants ($p<0.001$). Bony enlargement was observed in 71 (59.2%; 95% CI: 50.2%-67.6%) patients and was also significantly more frequent than the 50% reference proportion ($z=2.01$; $p=0.044$).

Knee swelling or effusion was present in 46 (38.3%; 95% CI: 30.1%-47.3%) patients, whereas varus or valgus deformity was identified in 37 (30.8%; 95% CI: 23.3%-39.6%). These findings occurred in significantly fewer than half of the patients, with $p=0.011$ and $p<0.001$, respectively. Thus, crepitus and joint-line tenderness were the predominant clinical findings, whereas fixed deformity was comparatively less common.

The mean WOMAC pain score was 11.20±4.20 out of 20, corresponding to 56.0% of the maximum possible score. The mean stiffness score was 3.27±1.60 out of 8, representing 40.9% of the maximum score. The mean physical-function score was 39.86±15.10 out of 68, equivalent to 58.6%, while the mean total WOMAC score was 48.80±17.20 out of 96, or 50.8% of the maximum score. Physical-function impairment therefore represented the most prominent WOMAC domain after standardisation.

Based on the total WOMAC score, 31 (25.8%; 95% CI: 18.8%-34.3%) patients had mild impairment, 47 (39.2%; 95% CI: 30.9%-48.1%) had moderate impairment and 42 (35.0%; 95% CI: 27.0%-43.9%) had severe impairment. The distribution across these three categories was not statistically different from an equal distribution ($\chi^2=3.95$, df=2; $p=0.139$). Nevertheless, 89 (74.2%) patients collectively had moderate or severe functional impairment, demonstrating a considerable burden of disability in the study population.

Table 4: Association of BMI categories with pain, stiffness, physical-function impairment and overall WOMAC score (N=120)

Outcome	Normal weight (n=27), Mean (SD)	Overweight (n=34), Mean (SD)	Obesity class I (n=38), Mean (SD)	Obesity class II (n=21), Mean (SD)	ANOVA F	P value
Numerical pain-rating score, 0-10	3.80 (1.50)	5.10 (1.60)	6.40 (1.70)	7.30 (1.40)	24.21	<0.001*
WOMAC pain score, 0-20	7.10 (3.10)	9.80 (3.50)	12.90 (3.70)	15.00 (3.30)	26.87	<0.001*
WOMAC stiffness score, 0-8	2.10 (1.20)	2.90 (1.30)	3.80 (1.50)	4.40 (1.40)	14.18	<0.001*
WOMAC physical-function score, 0-68	24.60 (9.10)	34.80 (10.40)	46.70 (11.80)	55.30 (10.90)	40.68	<0.001*
Overall WOMAC score, 0-96	30.50 (10.20)	42.80 (11.60)	57.10 (12.80)	67.00 (11.40)	48.53	<0.001*

Pairwise comparison of overall WOMAC scores

BMI comparison	Mean difference (95% CI)	Test statistic	Adjusted P value†
Overweight versus normal weight	12.30 (5.95-18.65)	$t=3.87$	<0.001*
Obesity class I versus normal weight	26.60 (20.37-32.83)	$t=8.52$	<0.001*
Obesity class II versus normal weight	36.50 (29.77-43.23)	$t=10.84$	<0.001*
Obesity class I versus overweight	14.30 (8.57-20.03)	$t=4.98$	<0.001*
Obesity class II versus overweight	24.20 (17.88-30.52)	$t=7.65$	<0.001*
Obesity class II versus obesity class I	9.90 (3.78-16.02)	$t=3.23$	0.009*

†Tukey-adjusted post-hoc comparison.

Categorical clinical outcomes according to BMI

Outcome	Normal weight (n=27), n (%)	Overweight (n=34), n (%)	Obesity class I (n=38), n (%)	Obesity class II (n=21), n (%)	Test of significance	P value
Moderate-to-severe pain	7 (25.9%)	15 (44.1%)	25 (65.8%)	17 (81.0%)	$\chi^2=18.11$	<0.001*
Severe functional impairment	4 (14.8%)	10 (29.4%)	20 (52.6%)	15 (71.4%)	$\chi^2=19.73$	<0.001*
Restricted knee movement	11 (40.7%)	20 (58.8%)	29 (76.3%)	19 (90.5%)	$\chi^2=16.71$	0.001*

**Statistically significant at p<0.05. All confidence intervals are 95% confidence intervals.*

Table 4 shows a clear increase in pain, stiffness and functional impairment across successive BMI categories. The mean numerical pain-rating score increased from 3.80±1.50 among normal-weight patients to 5.10±1.60 among overweight patients, 6.40±1.70 among patients with class I obesity and 7.30±1.40 among those with class II obesity. The difference across BMI categories was statistically significant (ANOVA $F=24.21$; $p<0.001$).

The WOMAC pain score increased progressively from 7.10±3.10 in the normal-weight group to 9.80±3.50 in the overweight group, 12.90±3.70 in the class I obesity group and 15.00±3.30 in the class II obesity group (F=26.87; p<0.001). A similar pattern was observed for WOMAC stiffness, with mean scores of 2.10±1.20, 2.90±1.30, 3.80±1.50 and 4.40±1.40, respectively (F=14.18; p<0.001).

Physical-function impairment showed a particularly strong relationship with BMI. The mean WOMAC physical-function score increased from 24.60±9.10 in normal-weight patients to 34.80±10.40 in overweight patients, 46.70±11.80 in patients with class I obesity and 55.30±10.90 in those with class II obesity (F=40.68; p<0.001). Similarly, the mean total WOMAC score increased from 30.50±10.20 in the normal-weight group to 42.80±11.60, 57.10±12.80 and 67.00±11.40 in the overweight, class I obesity and class II obesity groups, respectively. The overall difference was statistically significant (F=48.53; p<0.001).

Tukey-adjusted pairwise analysis demonstrated that the total WOMAC score was significantly higher in every higher BMI category. Compared with normal-weight patients, the score was higher by 12.30 points among overweight patients (95% CI: 5.95-18.65; p<0.001), by 26.60 points among patients with class I obesity (95% CI: 20.37-32.83; p<0.001) and by 36.50 points among those with class II obesity (95% CI: 29.77-43.23; p<0.001). Class I and class II obesity groups also had significantly higher scores than the overweight group. Furthermore, patients with class II obesity had a mean score 9.90 points higher than those with class I obesity (95% CI: 3.78-16.02; p=0.009).

The proportions with adverse clinical outcomes also rose with increasing BMI. Moderate-to-severe pain increased from 25.9% in normal-weight patients to 44.1% in overweight patients, 65.8% in patients with class I obesity and 81.0% in those with class II obesity (χ^2=18.11; p<0.001). Severe functional impairment increased correspondingly from 14.8% to 29.4%, 52.6% and 71.4%, respectively (χ^2=19.73; p<0.001). Restricted knee movement also increased from 40.7% among normal-weight patients to 90.5% among patients with class II obesity (χ^2=16.71; p=0.001).

Table 5: Baseline demographic, clinical, anthropometric, and radiographic characteristics of participants (N=120)

Baseline characteristic	**Mean (SD) or n (%)**	**95% CI**
Age, years	58.42 (9.61)	56.68–60.16
Age ≥60 years	53 (44.2%)	35.6%–53.1%
Female sex	73 (60.8%)	51.9%–69.1%
Male sex	47 (39.2%)	30.9%–48.1%
Duration of knee symptoms, years	4.63 (3.18)	4.06–5.20
Bilateral knee involvement	78 (65.0%)	56.1%–73.0%
Any comorbidity	51 (42.5%)	33.9%–51.4%
Hypertension	37 (30.8%)	23.3%–39.6%
Diabetes mellitus	22 (18.3%)	12.4%–26.2%
Height, cm	159.84 (8.72)	158.26–161.42
Weight, kg	67.91 (11.86)	65.77–70.05
BMI, kg/m²	26.48 (4.36)	25.69–27.27
Overweight or obese, BMI ≥23 kg/m²	93 (77.5%)	69.2%–84.1%
Numerical pain-rating score, 0–10	5.60 (2.00)	5.24–5.96
WOMAC total score, 0–96	48.80 (17.20)	45.69–51.91
Kellgren–Lawrence radiographic grade		
Grade II	31 (25.8%)	18.8%–34.3%
Grade III	54 (45.0%)	36.4%–53.9%
Grade IV	35 (29.2%)	21.8%–38.0%

BMI: body mass index; WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index; CI: confidence interval.

The mean age of the participants was 58.42±9.61 years, and females constituted 60.8% of the sample. The mean duration of knee symptoms was 4.63±3.18 years, while 65.0% had bilateral knee involvement and 42.5% had at least one comorbidity. The mean BMI was 26.48±4.36 kg/m², and 77.5% of participants were overweight or obese according to Asian Indian BMI criteria. Grade III was the most frequent Kellgren–Lawrence radiographic grade, observed in 45.0% of patients. The mean pain and total WOMAC scores were 5.60±2.00 and 48.80±17.20, respectively.

Table 6: Multiple linear regression analysis of factors associated with total WOMAC score (N=120)

Predictor	**Unstandardized β**	**SE**	**Standardized β**	**95% CI for β**	**t value**	**P value**
BMI, per 1 kg/m² increase	1.72	0.28	0.436	1.17–2.27	6.14	<0.001*
Age, per 1-year increase	0.11	0.07	0.105	−0.03–0.25	1.57	0.119
Female sex†	3.10	1.94	0.078	−0.74–6.94	1.60	0.113
Symptom duration, per 1-year increase	0.62	0.23	0.115	0.16–1.08	2.70	0.008*
Presence of any comorbidity‡	2.25	1.78	0.065	−1.28–5.78	1.26	0.209
Kellgren–Lawrence grade, per one-grade increase	5.46	0.91	0.281	3.66–7.26	6.00	<0.001*
Constant	−20.74	6.92	—	−34.45 to −7.03	−3.00	0.003*

Model summary: R^2=0.540; adjusted R^2=0.516; F(6,113)=22.11; p<0.001; Durbin–Watson statistic=1.91; maximum VIF=1.84.

Dependent variable: total WOMAC score, ranging from 0 to 96. Higher scores indicate greater clinical and functional impairment. β: regression coefficient; SE: standard error; CI: confidence interval; VIF: variance inflation factor. †Reference category: male sex. ‡Reference category: no comorbidity. *Statistically significant at p<0.05.

The multiple linear regression model was statistically significant and explained 54.0% of the variance in total WOMAC score. After adjustment for age, sex, symptom duration, comorbidity, and radiographic grade, BMI remained independently associated with functional severity. Each 1 kg/m² increase in BMI was associated with a 1.72-point increase in total WOMAC score (95% CI: 1.17–2.27; p<0.001). Longer symptom duration and higher Kellgren–Lawrence grade were also independent predictors of a higher WOMAC score. Age, sex, and comorbidity status were not independently significant. The maximum VIF was 1.84, indicating no important multicollinearity.

DISCUSSION

Association of BMI with clinical and functional severity

The present study demonstrated a clear progressive relationship between BMI and the clinical severity of knee osteoarthritis. Mean BMI increased from 23.60±2.90 kg/m² in patients with mild disease to 26.50±3.40 kg/m² in those with moderate disease and 29.10±3.80 kg/m² in those with severe disease. The overall difference was statistically significant (F=23.19; p<0.001). Patients with severe clinical disease had a mean BMI 5.50 kg/m² higher than those with mild disease. A comparable gradient was identified for functional severity: mean BMI increased from 23.70±2.80 kg/m² in mild impairment to 29.20±3.70 kg/m² in severe impairment (F=25.47; p<0.001).

These findings agree with Silverwood et al. (2015),[1] whose systematic review found that overweight and obesity were significant risk factors for the onset of knee pain and knee OA. The pooled odds of developing knee pain were 1.98 among overweight individuals and 2.66 among individuals with obesity. Similarly, Zheng et al. (2015),[2] in a meta-analysis of 14 prospective studies, reported that overweight and obesity increased the risk of knee OA by 2.45 and 4.55 times, respectively. Each 5 kg/m² increase in BMI was associated with an approximately 35% increase in knee-OA risk. Although these studies principally evaluated disease development rather than severity, their dose-response findings support the progressive association observed in the present study.

Reyes et al. (2016),[3] also reported a dose-dependent association between BMI and clinically diagnosed knee OA in a population-based cohort. The relationship was stronger for knee OA than for non-weight-bearing joints, emphasising the importance of mechanical loading. Excess body weight increases compressive forces across the tibiofemoral and patellofemoral compartments during walking, stair climbing and other weight-bearing activities. Repetitive loading may accelerate cartilage damage, subchondral bone remodelling and symptomatic progression.

The present study also found significant positive correlations between BMI and numerical pain score (r=0.52), WOMAC pain (r=0.49), stiffness (r=0.38), physical-function impairment (r=0.57) and total WOMAC score (r=0.58), with all p values <0.001. Raud et al. (2020),[4] reported similar results among patients with knee OA and excess weight. In their study, WOMAC function scores were progressively higher across obesity categories, and patients with class II/III obesity had significantly worse physical disability than overweight patients. They also recorded lower physical-activity levels and greater difficulty with activities of daily living in patients with more advanced obesity.

Gersing et al. (2016),[5] showed that overweight and obese adults who achieved greater weight loss experienced slower progression of cartilage degeneration and better clinical outcomes over 48 months. Their findings provide longitudinal support for the association observed in the present cross-sectional study. The stronger correlations of BMI with physical-function and total WOMAC scores than with stiffness suggest that excess body weight may primarily affect weight-bearing activities and overall disability.

Li et al. (2016),[6] observed that metabolic syndrome and its components aggravated pain and reduced knee function in people with OA. This indicates that BMI may represent both mechanical loading and a wider metabolic-inflammatory phenotype. Berenbaum et al. (2018),[7] further explained that obesity could influence OA through excessive mechanical stress, adipokine imbalance, low-grade systemic inflammation and altered subchondral bone metabolism. Therefore, the relationship between BMI and severity cannot be attributed exclusively to joint loading.

Distribution of BMI among patients with knee osteoarthritis

The mean BMI of the participants was 26.48±4.36 kg/m², significantly above the Asian Indian overweight threshold of 23.0 kg/m² (p<0.001). Only 22.5% had normal BMI, while 28.3% were overweight, 31.7% had class I obesity and 17.5% had class II obesity. Thus, 77.5% of the patients were either overweight or obese. This proportion was significantly greater than the reference proportion of 50% (p<0.001).

The predominance of excess body weight in the current study is consistent with the global epidemiological evidence. Long et al. (2022),[8] found that the prevalence and burden of knee OA increased markedly between 1990 and 2019, with increasing BMI identified as an important contributor to this trend. The relatively high prevalence of excess weight in the present study may also reflect the use of lower, ethnicity-appropriate BMI cut-offs for Asian Indian adults. Consequently, direct comparison with studies applying the conventional WHO overweight threshold of 25 kg/m² should be made cautiously.

Katz et al. (2021),[9] described obesity as one of the principal modifiable risk factors for symptomatic knee OA and noted that the burden of OA was expected to increase with ageing and rising obesity prevalence. The finding that class I obesity was the most common category in the present study may be clinically important because it identifies a substantial group in whom weight-management intervention remains feasible before progression to more severe obesity and disability.

Bannuru et al. (2019),[10] in the OARSI recommendations, identified structured exercise, education and dietary weight management as core treatments for knee OA in patients with excess weight. Kolasinski et al. (2020),[11] similarly recommended weight loss for overweight and obese patients with knee OA and reported a dose-response relationship, whereby greater weight reduction was generally associated with greater improvement in symptoms and function. These recommendations strengthen the clinical relevance of the high prevalence of overweight and obesity observed in the current population.

Clinical and functional severity

The mean duration of knee symptoms was 4.63±3.18 years, while the mean pain score was 5.60±2.00, indicating moderate pain. Crepitus was the most frequent clinical finding, occurring in 84.2% of patients, followed by joint-line tenderness in 80.8%, morning stiffness in 69.2%, restricted knee movement in 65.8% and bony enlargement in 59.2%. Knee swelling or effusion was present in 38.3%, while varus or valgus deformity was observed in 30.8%.

These findings are compatible with the recognised clinical spectrum of knee OA. Katz et al. (2021),[9] described activity-related pain, brief morning stiffness, crepitus, bony enlargement, tenderness and reduced movement as characteristic features. The relatively lower frequency of effusion and deformity in the present study may indicate that these findings are more closely associated with inflammatory exacerbations or structurally advanced disease, whereas crepitus and tenderness occur across a broader range of severity.

The mean WOMAC scores were 11.20±4.20 for pain, 3.27±1.60 for stiffness, 39.86±15.10 for physical function and 48.80±17.20 for the total score. After standardisation, physical-function impairment was the most affected domain at 58.6%, followed by pain at 56.0%. Overall, 39.2% of participants had moderate impairment and 35.0% had severe impairment; therefore, 74.2% had moderate-to-severe functional limitation.

Raud et al. (2020),[4] similarly reported a considerable functional burden among patients with knee OA and obesity. Their mean WOMAC function score increased from 36.2 among overweight patients to 45.6 among those with class II/III obesity. The greater functional impairment observed among patients with higher obesity grades was accompanied by lower walking and total physical-activity levels. These findings support the high burden of functional limitation identified in the present study.

Duong et al. (2023),[12] emphasised that knee-OA pain and disability are influenced by multiple interacting factors, including structural disease, muscle weakness, obesity, psychological factors, comorbidities and physical inactivity. Therefore, the moderate-to-severe WOMAC impairment found in the present study should not be interpreted as an effect of BMI alone. Nevertheless, the consistent association across pain, movement restriction and WOMAC domains indicates that BMI was an important marker of clinical burden.

The predominance of physical-function impairment may initiate a self-reinforcing cycle. Pain and restricted movement reduce physical activity and quadriceps strength, which may contribute to further weight gain and greater joint loading. This cycle is clinically important because both weight management and appropriately prescribed exercise may be required; dietary weight reduction without muscle-strengthening intervention may not adequately restore function.

Association of BMI categories with WOMAC domains

A strong dose-response pattern was observed across BMI categories. The mean numerical pain score increased from 3.80 in normal-weight patients to 7.30 in patients with class II obesity (F=24.21; p<0.001). WOMAC pain increased from 7.10 to 15.00 (F=26.87; p<0.001), stiffness from 2.10 to 4.40 (F=14.18; p<0.001), physical-function score from 24.60 to 55.30 (F=40.68; p<0.001) and total WOMAC score from 30.50 to 67.00 (F=48.53; p<0.001).

The total WOMAC score was significantly different between every pair of BMI categories. Compared with normal-weight patients, the adjusted mean difference was 12.30 points for overweight patients, 26.60 points for class I obesity and 36.50 points for class II obesity. Even class II obesity was associated with a score 9.90 points higher than class I obesity (p=0.009). This suggests that the association was not confined to a simple normal-weight versus obese comparison but continued across successive degrees of excess weight.

Raud et al. (2020),[4] reported a similar obesity-grade relationship with WOMAC physical disability and activity limitation. The present study demonstrated a larger separation between BMI categories, which could reflect differences in ethnicity, BMI thresholds, disease duration, radiographic severity, treatment exposure or comorbidity distribution.

Messier et al. (2018),[13] found that intentional weight loss among overweight and obese adults with symptomatic knee OA was associated with dose-dependent clinical benefits, with greater weight loss generally producing greater reductions in pain and improvements in function. Gersing et al. (2016),[5] also demonstrated that the extent of weight loss was related to slower cartilage degeneration and symptomatic improvement. These longitudinal findings provide biological and clinical support for the inverse interpretation of the present gradient: if increasing BMI is associated with worsening symptoms, meaningful weight reduction may improve at least part of this burden.

The categorical findings were consistent with the continuous scores. Moderate-to-severe pain increased from 25.9% in normal-weight patients to 81.0% among patients with class II obesity (p<0.001). Severe functional impairment increased from 14.8% to 71.4% (p<0.001), while restricted movement increased from 40.7% to 90.5% (p=0.001). These results suggest that higher BMI was associated not only with modest changes in average scores but also with clinically important adverse outcomes.

The findings are also consistent with contemporary clinical guidance. The National Institute for Health and Care Excellence (2022),[14] recommends therapeutic exercise and weight-management support for people with knee OA who are overweight or obese. The guideline notes that any weight loss is likely to be beneficial, although approximately 10% reduction in body weight may provide greater improvement than 5%. More recently, Bliddal et al. (2024),[15] demonstrated in a randomised trial that substantial pharmacologically assisted weight loss among people with obesity and knee OA was accompanied by greater reductions in knee pain than placebo, reinforcing the clinical connection between obesity reduction and symptomatic improvement.

Lv et al. (2024),[16] found significant associations between obesity indices and knee OA, suggesting that BMI remains useful but may not fully characterise obesity-related risk. Measures of central obesity and body-fat distribution may provide additional information because individuals with similar BMI can differ substantially in fat mass, muscle mass and metabolic risk. Future studies should therefore consider waist circumference, waist-to-height ratio, body composition and metabolic parameters alongside BMI.

Baseline characteristics

The baseline characteristics showed that the study population predominantly consisted of middle-aged and older adults, with a mean age of 58.42±9.61 years, and females constituted 60.8% of participants. This demographic pattern is consistent with Katz et al. (2021),[9] who reported that symptomatic knee osteoarthritis becomes increasingly common with advancing age and is more frequently encountered among women. Long et al. (2022),[8] also demonstrated a substantial age-related increase in the global prevalence of knee osteoarthritis, with a particularly high burden among older women. Bilateral knee involvement was present in 65.0% of participants, indicating a considerable overall disease burden. Furthermore, 42.5% had at least one comorbidity, including hypertension in 30.8% and diabetes mellitus in 18.3%. Li et al. (2016),[6] similarly observed that metabolic syndrome and its components were associated with more severe osteoarthritis pain, depressive symptoms and impaired knee function.

The mean BMI was 26.48±4.36 kg/m², and 77.5% of the participants were overweight or obese according to Asian Indian BMI criteria. These findings demonstrate that excess body weight was highly prevalent among patients with knee osteoarthritis. Raud et al. (2020),[4] similarly reported a high prevalence of overweight and obesity among patients with knee osteoarthritis and found that increasing obesity severity was associated with greater pain, physical disability and reduced physical activity. Zheng and Chen (2015),[2] in a systematic review and meta-analysis of prospective studies, found a dose-response relationship between BMI and the risk of developing knee osteoarthritis. Reyes et al. (2016),[3] also demonstrated that overweight and obesity were associated with a progressively increased risk of clinically diagnosed knee osteoarthritis. These observations support the role of excess body weight as an important modifiable factor in the occurrence and symptomatic burden of knee osteoarthritis.

The participants had a mean pain score of 5.60±2.00 and a mean total WOMAC score of 48.80±17.20, indicating moderate pain and substantial functional limitation. Nearly three-fourths of the participants had Kellgren–Lawrence grade III or IV disease, suggesting that a large proportion had moderate-to-advanced structural osteoarthritis. Katz et al. (2021),[9] described pain, stiffness, crepitus, reduced range of movement and functional impairment as the principal manifestations of symptomatic knee osteoarthritis. Duong et al. (2023),[12] further noted that knee-OA disability results from the interaction of structural damage, obesity, muscle weakness, comorbidities and physical inactivity. The coexistence of excess body weight, radiographic severity and functional limitation observed in the present study is therefore consistent with the multidimensional nature of knee-OA severity described in the literature.

Multiple linear regression analysis

In the multiple linear regression analysis, BMI remained an independent predictor of total WOMAC score after adjustment for age, sex, symptom duration, comorbidities and Kellgren–Lawrence grade. Each 1 kg/m² increase in BMI was associated with a 1.72-point increase in total

WOMAC score (95% CI: 1.17–2.27; p<0.001). The standardized regression coefficient for BMI was 0.436, indicating that BMI was the strongest independent predictor included in the model. This finding is consistent with Raud et al. (2020),[4] who demonstrated a direct relationship between the degree of obesity and the clinical and functional consequences of knee osteoarthritis. Their study showed that patients with higher obesity grades had greater WOMAC functional impairment and lower levels of physical activity. Lv et al. (2024),[16] likewise found significant associations between several obesity indices and knee osteoarthritis, supporting the importance of adiposity in the clinical expression of the disease.

The association between BMI and WOMAC severity may be explained through both mechanical and metabolic mechanisms. Greater body weight increases loading across the tibiofemoral and patellofemoral joints during standing, walking and stair climbing. Repeated excessive loading may accelerate cartilage degeneration and subchondral bone changes. Berenbaum et al. (2018),[7] additionally emphasised that adipose tissue produces adipokines and inflammatory mediators that may contribute to synovial inflammation, cartilage catabolism and pain sensitisation. Therefore, the independent association observed after adjustment for radiographic grade suggests that the effect of obesity on symptoms and disability may not be explained solely by structural joint damage.

Longer symptom duration was another independent predictor of higher total WOMAC score (β=0.62; 95% CI: 0.16–1.08; p=0.008). This finding suggests that prolonged exposure to pain, stiffness, muscle weakness and reduced physical activity may progressively worsen functional capacity. Duong et al. (2023),[12] described a reciprocal relationship in which knee pain reduces physical activity and muscle strength, thereby increasing disability and potentially promoting further weight gain. This may create a self-perpetuating cycle of pain, inactivity, obesity and declining function. Early interventions directed towards exercise, muscle strengthening and weight control may therefore help to interrupt this cycle.

Kellgren–Lawrence grade was also independently associated with the total WOMAC score. Each one-grade increase was associated with a 5.46-point increase in the WOMAC score (95% CI: 3.66–7.26; p<0.001). This indicates that more advanced radiographic disease was accompanied by greater symptomatic and functional impairment, even after controlling for BMI and other clinical variables. Nevertheless, radiographic abnormalities do not always correspond directly with the intensity of pain or disability. Katz et al. (2021),[9] and Duong et al. (2023),[12] noted that the relationship between structural severity and symptoms is influenced by multiple factors, including obesity, muscle weakness, inflammation, psychological status and pain sensitisation. The independent effects of both BMI and radiographic grade in the present model support the importance of assessing structural and patient-reported severity together.

Age, sex and the presence of comorbidities were not statistically significant after adjustment, although they may still influence knee-OA symptoms indirectly. The absence of independent associations may indicate that their crude effects were partly explained by BMI, disease duration or radiographic severity. Silverwood et al. (2015),[1] identified age, female sex, previous knee injury and excess body weight as important risk factors for knee osteoarthritis; however, factors associated with the development of OA may not necessarily remain independent predictors of symptom severity after the disease has become established. Similarly, the effect of comorbidities may vary according to their number, severity and level of control, which were not separately evaluated in the present analysis.

The regression model explained 54.0% of the variability in total WOMAC score, indicating good explanatory ability for a clinical cross-sectional model. However, approximately 46% of the variability remained unexplained and may be attributable to muscle strength, physical activity, joint alignment, central adiposity, analgesic use, psychological factors and pain sensitisation. Nevertheless, the findings provide clinical support for recommendations by Bannuru et al. (2019),[10] Kolasinski et al. (2020),[11] and the National Institute for Health and Care Excellence (2022),[14] which identify exercise and weight management as core interventions for overweight or obese patients with knee osteoarthritis. Messier et al. (2018),[13] and Gersing et al. (2016),[5] further demonstrated that greater intentional weight loss was associated with improvements in symptoms and function and with slower cartilage degeneration. More recently, Bliddal et al. (2024),[15] reported that substantial weight reduction among patients with obesity and knee osteoarthritis was accompanied by meaningful improvement in knee pain. Collectively, these studies support the present finding that BMI is an important, potentially modifiable determinant of clinical and functional severity in knee osteoarthritis.

CONCLUSION

The study demonstrated a significant association between increasing body mass index and worsening clinical and functional severity of knee osteoarthritis. Mean BMI increased progressively from patients with mild clinical disease to those with severe disease. BMI showed significant positive correlations with pain, stiffness, physical-function impairment and total WOMAC score, with the strongest association observed for overall functional disability. Patients with class II obesity had the highest pain and WOMAC scores and the greatest prevalence of restricted knee movement and severe functional impairment. These findings indicate that excess body weight is an important modifiable factor associated with the symptomatic and functional burden of knee osteoarthritis. Incorporating weight assessment, weight-management counselling, therapeutic exercise and rehabilitation into routine knee-OA care may help reduce pain and improve functional capacity. However, the cross-sectional design establishes association rather than causation.

Limitations

1. The cross-sectional design did not establish a temporal or causal relationship between increased BMI and knee-OA severity. Severe pain and disability could themselves reduce physical activity and contribute to weight gain.
2. The study was conducted at a single hospital, which may limit the generalisability of the findings to the wider community or other healthcare settings.
3. The relatively modest sample size of 120 patients may have limited the precision of subgroup estimates, particularly for patients with class II obesity.
4. Participants were recruited from hospital services and may have had more severe symptoms than patients with knee OA managed in primary care or those not seeking treatment, resulting in selection bias.
5. BMI does not distinguish between fat mass and lean muscle mass and does not describe central adiposity. Waist circumference, waist-to-height ratio and body-composition measurements were not assessed.
6. Pain and functional impairment were evaluated partly using patient-reported measures and were therefore subject to recall bias, reporting bias and individual differences in pain perception.
7. Potential confounding factors such as age, sex, occupation, socioeconomic status, physical activity, muscle strength, dietary habits, analgesic use, comorbidities and psychological status might have influenced the observed relationships.
8. Radiographic severity and joint alignment were not fully incorporated into every analysis. Consequently, the independent contribution of BMI after controlling for structural severity could not be determined comprehensively.
9. The study did not measure inflammatory markers, adipokines or metabolic parameters that could clarify the biological pathways linking obesity with knee-OA symptoms.
10. Information on previous weight changes and duration of overweight or obesity was unavailable; therefore, the effects of cumulative exposure to excess body weight could not be examined.
11. The use of Asian Indian BMI cut-offs may limit direct comparison with international studies using conventional WHO categories.
12. Longitudinal follow-up was not performed, so the influence of BMI on disease progression or changes in pain and function over time could not be assessed.

REFERENCES

1. Silverwood V, Blagojevic-Bucknall M, Jinks C, Jordan JL, Protheroe J, Jordan KP. Current evidence on risk factors for knee osteoarthritis in older adults: a systematic review and meta-analysis. Osteoarthritis Cartilage. 2015;23(4):507-15.
2. Zheng H, Chen C. Body mass index and risk of knee osteoarthritis: systematic review and meta-analysis of prospective studies. BMJ Open. 2015;5(12):e007568.
3. Reyes C, Leyland KM, Peat G, Cooper C, Arden NK, Prieto-Alhambra D. Association between overweight and obesity and risk of clinically diagnosed knee, hip, and hand osteoarthritis: a population-based cohort study. Arthritis Rheumatol. 2016;68(8):1869-75.
4. Raud B, Gay C, Guiguet-Auclair C, Bonnin A, Gerbaud L, Pereira B, et al. Level of obesity is directly associated with the clinical and functional consequences of knee osteoarthritis. Sci Rep. 2020;10(1):3601.
5. Gersing AS, Schwaiger BJ, Nevitt MC, Zarnowski J, Joseph GB, Feuerriegel G, et al. Progression of cartilage degeneration and clinical symptoms in obese and overweight individuals is dependent on the amount of weight loss: 48-month data from the Osteoarthritis Initiative. Osteoarthritis Cartilage. 2016;24(7):1126-34.
6. Li H, George DM, Jaarsma RL, Mao X. Metabolic syndrome and components exacerbate osteoarthritis symptoms of pain, depression and reduced knee function. Ann Transl Med. 2016;4(7):133.
7. Berenbaum F, Wallace IJ, Lieberman DE, Felson DT. Modern-day environmental factors in the pathogenesis of osteoarthritis. Nat Rev Rheumatol. 2018;14(11):674-81.
8. Long H, Liu Q, Yin H, Diao N, Zhang Y, Lin J, et al. Prevalence trends of site-specific osteoarthritis from 1990 to 2019: findings from the Global Burden of Disease Study 2019. Arthritis Rheumatol. 2022;74(7):1172-83.
9. Katz JN, Arant KR, Loeser RF. Diagnosis and treatment of hip and knee osteoarthritis: a review. JAMA. 2021;325(6):568-78.
10. Bannuru RR, Osani MC, Vaysbrot EE, Arden NK, Bennell K, Bierma-Zeinstra SMA, et al. OARSI guidelines for the non-surgical management of knee, hip, and polyarticular osteoarthritis. Osteoarthritis Cartilage. 2019;27(11):1578-89.
11. Kolasinski SL, Neogi T, Hochberg MC, Oatis C, Guyatt G, Block J, et al. 2019 American College of Rheumatology/Arthritis Foundation guideline for the management of osteoarthritis of the hand, hip, and knee. Arthritis Care Res (Hoboken). 2020;72(2):149-62.
12. Duong V, Oo WM, Ding C, Culvenor AG, Hunter DJ. Evaluation and treatment of knee pain: a review. JAMA. 2023;330(16):1568-80.
13. Messier SP, Resnik AE, Beavers DP, Mihalko SL, Miller GD, Nicklas BJ, et al. Intentional weight loss in overweight and obese patients with knee osteoarthritis: is more better? Arthritis Care Res (Hoboken). 2018;70(11):1569-75.
14. National Institute for Health and Care Excellence. Osteoarthritis in over 16s: diagnosis and management. NICE guideline NG226. London: NICE; 2022.
15. Bliddal H, Bays H, Czernichow S, Uddén Hemmingsson J, Hjelmesæth J, Hoffmann Morville T, et al. Once-weekly semaglutide in persons with obesity and knee osteoarthritis. N Engl J Med. 2024;391(17):1573-83.
16. Lv H, Zhang L, Guo W, Zhang Y, Wang J, Liu Y, et al. Association between obesity measurement indexes and prevalence of knee osteoarthritis: a cross-sectional study. BMC Musculoskelet Disord. 2024;25:879.