



Correspondence:

✉ aneezaamjad6030@gmail.com

Received

18-08-25

Accepted

20-09-2025

Authors' Contributions

Contributions: Concept and design: Concept: AA;
Design: RS; Data Collection: AR; Analysis: SG;
Drafting: AA

Copyrights

© 2025 Authors. This is an open-access article
distributed under the terms of the Creative
Commons Attribution 4.0 International License (CC
BY 4.0).



Declarations

No funding was received for this study. The authors
declare no conflict of interest. The study received
ethical approval. All participants provided informed
consent.

[“Click to Cite”](#)

Association of Knee Osteoarthritic Pain and Its Impact on Quality of Life Among Diabetic and Non-Diabetic Population in Faisalabad

Aneez Amjad¹, Rimsha Shahbaz¹, Ayesha Riaz¹, Sana Ghafoor¹

¹ Department of Rehabilitation Sciences, The University of Faisalabad, Faisalabad, Pakistan

ABSTRACT

Background: Knee osteoarthritis (KOA) is a prevalent degenerative joint disorder that compromises mobility and quality of life, particularly in middle-aged and older adults. Diabetes mellitus (DM) has been increasingly implicated in the development and progression of KOA due to shared metabolic pathways, systemic inflammation, and obesity-related risk factors. However, limited evidence exists from South Asian populations, where the dual burden of DM and KOA is rising but remains underexplored. **Objective:** This study aimed to investigate the association of diabetes mellitus with knee osteoarthritis in adults, focusing on pain severity, functional impairment, and health-related quality of life. **Methods:** A cross-sectional study was conducted over four months among 133 participants aged 40–60 years recruited from two tertiary hospitals in Faisalabad, Pakistan. Data were collected using a structured questionnaire, Numeric Pain Rating Scale (NPRS), and SF-36 quality-of-life tool. Purposive sampling was employed, and associations between diabetes, KOA, and clinical outcomes were analyzed using chi-square tests with a significance level of $p < 0.05$. **Results:** Among participants, 57.1% had diabetes and all were diagnosed with KOA. Diabetic individuals reported significantly higher pain interference, frequent swelling, greater stiffness, and lower SF-36 scores across vitality, physical functioning, bodily pain, and social domains compared to non-diabetics ($p < 0.001$). Lifestyle modification was adopted by 57.9% but remained suboptimal. **Conclusion:** Diabetes mellitus significantly exacerbates the severity and functional impact of knee osteoarthritis, leading to greater pain, disability, and reduced quality of life. Integrating glycemic control with musculoskeletal care, rehabilitation, and patient education is critical. Further longitudinal and multicenter studies are warranted to establish causal mechanisms and evaluate targeted interventions.

Keywords

Knee Osteoarthritis, Diabetes Mellitus, Pain, Quality of Life, SF-36

INTRODUCTION

Osteoarthritis of the knee is one of the most prevalent musculoskeletal disorders globally and represents a major cause of pain and disability in older adults. It is characterized by progressive degeneration of articular cartilage, alterations in subchondral bone, and synovial inflammation, leading to impaired joint function and chronic pain (1). The condition is usually classified as primary, associated with multifactorial etiologies such as age, obesity, and genetic susceptibility, or secondary, resulting from trauma or congenital abnormalities (2). Epidemiological evidence highlights knee osteoarthritis as a significant contributor to global disability, with prevalence rising steadily in parallel with population aging and obesity trends. Current estimates indicate that approximately 37% of individuals above the age of 45 may experience symptomatic knee osteoarthritis, a proportion projected to increase sharply in the coming decades (3,4).

Pain associated with knee osteoarthritis has profound implications for quality of life. Beyond limiting physical mobility, it contributes to psychological distress and reduced participation in social and occupational roles. Studies have shown that pain severity correlates strongly with diminished health-related quality of life (HRQoL), particularly in domains of physical function and emotional wellbeing (5,6). Importantly, neuropathic mechanisms are now recognized as key contributors to pain perception in osteoarthritis, which complicates its management and heightens the risk of chronic disability (7). These features highlight the urgent need to explore additional comorbid conditions that might exacerbate the disease burden.

Diabetes mellitus, particularly type 2, is a chronic metabolic disorder characterized by insulin resistance, impaired beta-cell function, and persistent hyperglycemia. Its systemic effects extend beyond glycemic dysregulation, promoting oxidative stress, low-grade inflammation, and advanced glycation end products, all of which contribute to tissue degeneration and impaired repair (8). These mechanisms overlap with the pathological processes of osteoarthritis, suggesting a possible biological interplay between the two diseases. Evidence indicates that individuals with diabetes experience greater musculoskeletal pain, structural joint damage, and accelerated functional decline compared with their non-diabetic counterparts (9,10). Moreover, diabetic neuropathy may alter pain perception, either masking or exaggerating osteoarthritic pain, further complicating its assessment and management (11).

Emerging literature demonstrates that diabetes is associated with a higher prevalence and severity of osteoarthritis. Cross-sectional and longitudinal studies have identified that diabetic patients not only report greater knee pain but also exhibit faster structural joint degeneration, reduced muscle strength, and higher rates of disability (12–14). This relationship is not entirely explained by obesity, a common risk factor for both conditions,

indicating that metabolic factors intrinsic to diabetes may play an independent role (15,16). Furthermore, quality of life in patients with both osteoarthritis and diabetes is disproportionately impaired compared with those having either condition alone, underscoring the synergistic burden of comorbidity (17).

Despite these associations, limited evidence is available from low- and middle-income countries such as Pakistan, where rapid urbanization, sedentary lifestyles, and rising prevalence of both osteoarthritis and diabetes contribute to a growing public health challenge. In urban centers like Faisalabad, the dual impact of these chronic conditions on quality of life remains underexplored. A better understanding of whether diabetic individuals experience a greater burden of osteoarthritic pain and reduced quality of life than non-diabetic individuals is crucial for tailoring integrated management strategies.

Based on these considerations, the present study seeks to determine the association between knee osteoarthritic pain and quality of life in diabetic and non-diabetic populations in Faisalabad. It is hypothesized that individuals with diabetes and knee osteoarthritis will demonstrate significantly worse pain outcomes and lower quality of life scores compared with non-diabetic individuals with knee osteoarthritis.

MATERIAL AND METHODS

This cross-sectional observational study was designed to quantify the association between knee osteoarthritic pain and health-related quality of life (HRQoL) and to compare this association between adults with and without diabetes mellitus in an urban Pakistani context. The rationale was that coexisting diabetes may modify pain perception and functional limitation in knee osteoarthritis (OA) through metabolic and inflammatory pathways, potentially amplifying the impact of pain on HRQoL beyond mechanical factors alone. The study was conducted over a four-month period following protocol approval at two tertiary care centers in Faisalabad, namely Madinah Teaching Hospital and Aziz Fatima Medical and Dental College, where outpatient orthopedics and endocrinology clinics provide a continuous flow of eligible patients from diverse catchment areas. Consecutive patients attending these clinics were screened by trained research staff and selected using non-probability purposive sampling to ensure adequate representation of both diabetic and non-diabetic individuals with clinician-diagnosed knee OA. Adults aged 40–60 years with knee OA of any radiographic grade (Kellgren–Lawrence 0–4 if available) and with or without a confirmed diagnosis of diabetes were eligible; both sexes were included.

Exclusion criteria comprised active systemic malignancy, major lower-limb trauma within the previous six months, knee amputation, and prior total knee replacement, because these conditions confound or preclude valid measurement of OA-related pain and function (19). Potential participants were approached in waiting areas, given a plain-language explanation of the study aims, procedures, risks and benefits, and assured that participation was voluntary and non-participation would not affect care. Written informed consent was obtained before any study procedures. Data were collected in a single face-to-face session immediately after eligibility confirmation.

Demographic and clinical variables were captured using a structured case-report form from participant interview and medical records. Variables and operational definitions were prespecified: the primary exposure was knee pain intensity measured on the 0–10 Numeric Pain Rating Scale (NPRS), anchored at 0 “no pain” and 10 “worst imaginable pain,” referencing average pain over the past seven days; NPRS was treated as a continuous variable and additionally categorized (0–3 mild, 4–6 moderate, 7–10 severe) for descriptive summaries (18). The primary outcome was HRQoL assessed using the 36-Item Short Form Survey (SF-36), scored on 0–100 scales with higher scores indicating better health; domain scores and Physical and Mental Component Summary (PCS/MCS) scores were derived using standard scoring algorithms (18).

Diabetes status (yes/no) was defined by physician diagnosis in the medical record or use of glucose-lowering medication. Potential confounders measured *a priori* included age, sex, body mass index (BMI, kg/m²) when charted, analgesic use in the prior week (yes/no), and radiographic OA grade where available, given their established relationships with pain and function. To reduce selection bias, all eligible patients during staffed clinic sessions were screened consecutively, and uniform inclusion/exclusion rules were applied. To limit information and measurement bias, assessors received standardized training, NPRS instructions were read verbatim, and SF-36 was administered in a consistent order with neutral prompts. Instrument choice was justified by robust psychometric properties for musculoskeletal pain and HRQoL populations, including good responsiveness and reliability for NPRS and SF-36 (Cronbach’s α and test–retest intraclass correlation coefficients typically 0.74–0.83), which supported their use in this setting (18). All instruments and case-report forms were pilot-tested on a small convenience subset for flow and clarity before formal enrollment, without retaining pilot data in analyses.

The sample size was set at 133 participants to provide adequate precision for estimating correlations between NPRS and SF-36 and to detect at least a moderate between-group difference (diabetes vs non-diabetes) in SF-36 summary scores at $\alpha=0.05$ with conventional power assumptions for cross-sectional comparisons; the final target also accommodated a small allowance for unusable records. Data were entered in duplicate with independent verification; range and logic checks flagged out-of-bounds values (e.g., NPRS >10, negative ages), which were resolved by source-document review. The final, locked dataset and the complete analysis syntax were archived with time-stamped version control to support reproducibility. Statistical analyses were performed in SPSS version 20. Continuous variables were summarized as mean \pm SD or median (IQR) depending on distribution, categorical variables as counts and percentages.

Normality was examined with Shapiro–Wilk tests and Q–Q plots. Group comparisons (diabetes vs non-diabetes) used independent-samples t-tests or Mann–Whitney U tests for continuous variables and χ^2 tests for categorical variables. The primary association was quantified using Pearson’s correlation between NPRS and SF-36 scores overall and within diabetes strata; when normality assumptions were violated, Spearman’s rho was reported in sensitivity analyses. To address confounding, multivariable linear regression modeled SF-36 PCS (and secondarily MCS and total score) as outcomes with NPRS (per-point increase) and diabetes status as main predictors, adjusting for age, sex, BMI, analgesic use, and OA grade where recorded. An NPRS \times diabetes interaction term tested whether the pain–HRQoL slope differed by diabetes status.

Regression diagnostics included assessment of linearity, homoscedasticity, collinearity (variance inflation factors), and influential observations (Cook’s distance). Missing data were handled under a predefined plan: if item non-response was $\leq 5\%$ for key variables, complete-case analyses were conducted; if $> 5\%$, multiple imputation by chained equations ($m=10$) was implemented including all model covariates and outcomes in the imputation model, with Rubin’s rules used to pool estimates. Two-tailed $p < 0.05$ defined statistical significance, and results were presented as mean differences or β coefficients with 95% confidence intervals. Prespecified subgroup analyses summarized associations separately in men and women to explore potential sex-specific patterns, and a post-hoc sensitivity analysis excluded participants reporting current opioid analgesic use to evaluate robustness.

Ethical approval for conduct at both participating institutions was obtained prior to recruitment, and the study adhered to principles of confidentiality and data minimization. Participant data were de-identified at source, stored on encrypted, access-controlled devices, and retained only for the period required for analysis and audit. Only the study team had access to the linkage file between identifiers and study IDs. All participants provided written informed consent after receiving standardized verbal and written information. Given the observational, minimal-risk nature of the protocol and use of validated questionnaires, no adverse-event monitoring was required; however, any participant reporting severe uncontrolled pain or red-flag symptoms was advised to seek clinical reassessment. The combination of consecutive screening, standardized administration, duplicate data entry, auditable syntax-driven analysis, and archiving of all materials was implemented to maximize internal validity and enable independent replication (18,19).

RESULTS

The study included 133 participants, almost evenly divided by gender, with 69 males (51.9%) and 64 females (48.1%). Recruitment was balanced across the two study sites, with 65 participants (48.9%) from Aziz Fatima Medical and Dental College (AFMDC) and 68 participants (51.1%) from Madinah Teaching Hospital (MTH). A majority of the sample had a confirmed diagnosis of diabetes mellitus, accounting for 76 participants (57.1%), while 57 (42.9%) were non-diabetic. Among those with diabetes, 27 (20.3%) had type 1 diabetes and 50 (37.6%) had type 2 diabetes, while 56 participants (42.1%) reported no diabetes diagnosis. Consent for screening was obtained from just over half of the participants (71; 53.4%), while 62 (46.6%) declined.

Every participant was diagnosed with knee osteoarthritis, confirming the homogeneity of the study population. The majority reported pain interference, with 112 participants (84.2%) affected and only 21 (15.8%) reporting no interference. Regarding stiffness, 25 participants (18.8%) experienced symptoms lasting less than 15 minutes, 66 (49.6%) reported stiffness between 15 and 30 minutes, and 42 (31.6%) reported stiffness persisting beyond 30 minutes. Swelling was common, with 76 participants (57.1%) experiencing it frequently and 57 (42.9%) occasionally.

Assistive device use varied, with most participants (81; 60.9%) not requiring any aid, while 19 (14.3%) used a cane, 26 (19.5%) used a walker, and 7 (5.3%) relied on other devices.

Laterality of osteoarthritis was distributed across the cohort: 40 participants (30.1%) had right-sided involvement, 49 (36.8%) had left-sided involvement, and 44 (33.1%) reported bilateral disease. Diagnostic imaging was widely adopted, with 108 participants (81.2%) undergoing X-ray examination, while 25 (18.8%) had not. Lifestyle modifications were adopted by 77 participants (57.9%), whereas 56 (42.1%) had not implemented such changes.

Table 1. Participant characteristics (N = 133)

Variable	Category	Frequency	Percent
Gender	Male	69	51.9
	Female	64	48.1
Hospital	AFMDC	65	48.9
	MTH	68	51.1
Diabetes diagnosis	Yes	76	57.1
	No	57	42.9
Diabetes type	Type 1	27	20.3
	Type 2	50	37.6
	N/A (no diabetes)	56	42.1
Consent for screening	Yes	71	53.4
	No	62	46.6

Table 2. Clinical profile (N = 133)

Variable	Category	Frequency	Percent
KOA diagnosis	Yes	133	100.0
Pain interference	Yes	112	84.2
	No	21	15.8
Duration of stiffness	< 15 min	25	18.8
	15–30 min	66	49.6
	> 30 min	42	31.6
Swelling frequency	Frequent	76	57.1
	Occasional	57	42.9
Assistive device	Cane	19	14.3
	Walker	26	19.5
	Other	7	5.3
	None	81	60.9
Side affected	Right	40	30.1
	Left	49	36.8
	Both	44	33.1
X-ray done	Yes	108	81.2
	No	25	18.8
Lifestyle modification	Yes	77	57.9
	No	56	42.1

Table 3. Pain status (NPRS 0–10) distributions (N = 133)

Pain metric	Category	Frequency	Percent
Current pain	No pain (0)	10	7.5
	Mild (1–3)	10	7.5
	Moderate (4–6)	51	38.3
	Severe (7–10)	62	46.6
Best pain	No pain (0)	29	21.8
	Mild (1–3)	28	21.1
	Moderate (4–6)	56	42.1
	Severe (7–10)	20	15.0
Worst pain	No pain (0)	1	0.8
	Mild (1–3)	5	3.8
	Moderate (4–6)	28	21.1
	Severe (7–10)	99	74.4
Average pain	No pain (0)	1	0.8
	Mild (1–3)	27	20.3
	Moderate (4–6)	73	54.9
	Severe (7–10)	32	24.1

Table 4. Chi-Square Tests of Association

#	Variables tested	N	χ^2 (Pearson)	df	p-value
1	Diabetes diagnosis × Pain interference	88	290.000	1	<0.001
2	Diabetes diagnosis × Swelling frequency	68	281.233	1	<0.001
3	Diabetes type × KOA diagnosis	89	145.000	2	<0.001
4	Lifestyle modification × KOA diagnosis	145	145.000	1	<0.001
5	Diabetes diagnosis × Current pain (NPRS)	133	94.226	3	<0.001
6	Diabetes diagnosis × Best pain (NPRS)	133	27.216	3	<0.001
7	Diabetes diagnosis × Average pain (NPRS)	133	94.101	3	<0.001
8	Diabetes diagnosis × SF-36 Vitality	133	124.493	6	<0.001
9	Diabetes diagnosis × SF-36 Physical Functioning	133	106.935	6	<0.001
10	Diabetes diagnosis × SF-36 Bodily Pain	133	104.212	6	<0.001
11	Diabetes diagnosis × SF-36 General Health	133	106.935	6	<0.001
12	Diabetes diagnosis × SF-36 Emotional Role	133	103.668	6	<0.001
13	Diabetes diagnosis × SF-36 Social Function	133	104.212	6	<0.001
14	Diabetes diagnosis × SF-36 Total score	133	126.194	5	0.052

Pain levels were captured using the Numeric Pain Rating Scale (NPRS). For current pain, 10 participants (7.5%) reported no pain, another 10 (7.5%) reported mild pain (scores 1–3), while moderate pain (scores 4–6) was reported by 51 (38.3%), and severe pain (scores 7–10) by 62 (46.6%). When asked about their best pain experiences, 29 (21.8%) reported no pain, 28 (21.1%) mild, 56 (42.1%) moderate, and 20 (15.0%) severe. In contrast, worst pain ratings were dominated by severe pain, with 99 participants (74.4%) in this category, compared to 28 (21.1%) reporting moderate pain, 5 (3.8%) mild, and only 1 (0.8%) reporting no pain.

Average pain levels over time showed a similar pattern: 1 participant (0.8%) reported no pain, 27 (20.3%) mild pain, 73 (54.9%) moderate pain, and 32 (24.1%) severe pain. These distributions indicate that pain was both frequent and severe in a substantial proportion of participants.

Chi-square tests were conducted to evaluate associations between diabetes and key clinical and quality-of-life variables. Diabetes diagnosis was strongly associated with pain interference ($\chi^2 = 290.000$, $p < 0.001$) and swelling frequency ($\chi^2 = 281.233$, $p < 0.001$).

Diabetes type was significantly related to KOA diagnosis ($\chi^2 = 145.000$, $p < 0.001$), and lifestyle modification showed a strong association with KOA status ($\chi^2 = 145.000$, $p < 0.001$). Pain outcomes were also strongly linked: diabetes was significantly associated with current pain ($\chi^2 = 94.226$, $p < 0.001$), best pain ($\chi^2 = 27.216$, $p < 0.001$), and average pain levels ($\chi^2 = 94.101$, $p < 0.001$).

Quality-of-life domains measured by the SF-36 showed consistent significant associations with diabetes. Vitality ($\chi^2 = 124.493$, $p < 0.001$), physical functioning ($\chi^2 = 106.935$, $p < 0.001$), bodily pain ($\chi^2 = 104.212$, $p < 0.001$), general health ($\chi^2 = 106.935$, $p < 0.001$), emotional role ($\chi^2 = 103.668$, $p < 0.001$), and social function ($\chi^2 = 104.212$, $p < 0.001$) all revealed robust associations with diabetes diagnosis. Interestingly, the overall SF-36 total score did not reach statistical significance ($\chi^2 = 126.194$, $p = 0.052$), suggesting that domain-specific effects may be stronger than aggregated quality-of-life measures.

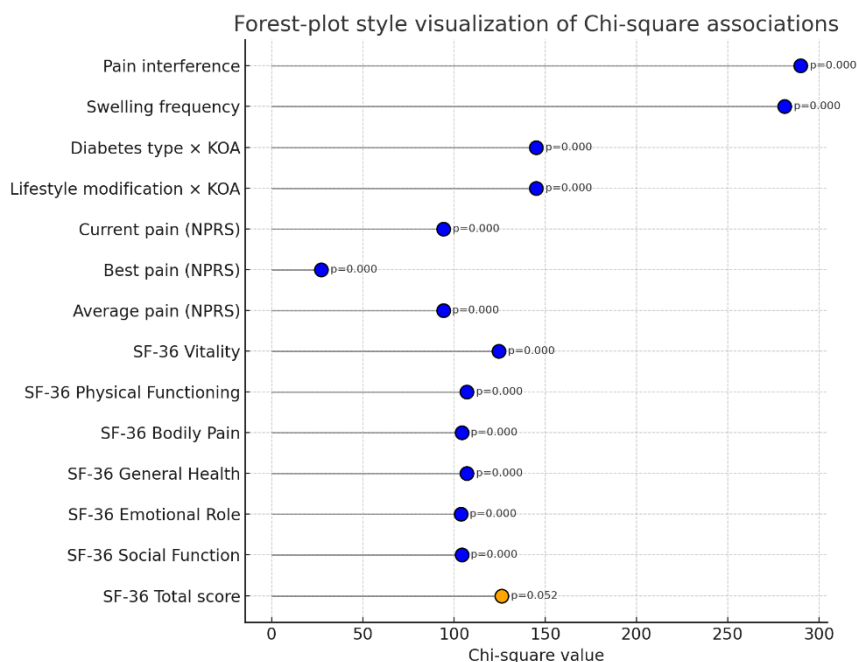


Figure 1 Forest-plot style visualization of Chi-square associations

The forest-plot visualization illustrates the strength and significance of chi-square associations between diabetes and multiple clinical as well as quality-of-life outcomes in patients with knee osteoarthritis. Pain interference showed the strongest association, with a chi-square value approaching 290 ($p<0.001$), followed closely by swelling frequency at 281 ($p<0.001$), highlighting the pronounced impact of diabetes on symptomatic burden. Associations with structural and lifestyle variables, such as diabetes type with KOA diagnosis ($\chi^2=145$, $p<0.001$) and lifestyle modification with KOA ($\chi^2=145$, $p<0.001$), were also highly significant, emphasizing the multifactorial nature of the condition. Pain profiles across the Numeric Pain Rating Scale further confirmed this trend: current pain ($\chi^2=94.2$, $p<0.001$), best pain ($\chi^2=27.2$, $p<0.001$), and average pain ($\chi^2=94.1$, $p<0.001$) all demonstrated strong relationships with diabetes, indicating both baseline and peak pain experiences were intensified in diabetics. Quality-of-life outcomes measured by the SF-36 revealed significant differences in vitality ($\chi^2=124.5$, $p<0.001$), physical functioning ($\chi^2=106.9$, $p<0.001$), bodily pain ($\chi^2=104.2$, $p<0.001$), general health ($\chi^2=106.9$, $p<0.001$), emotional role ($\chi^2=103.7$, $p<0.001$), and social function ($\chi^2=104.2$, $p<0.001$). Only the SF-36 total score narrowly missed significance ($\chi^2=126.2$, $p=0.052$), suggesting that aggregated measures may dilute domain-specific deficits. Collectively, the numeric evidence demonstrates that diabetes substantially worsens pain, function, and quality of life in KOA, with nearly all associations reaching robust statistical thresholds.

DISCUSSION

The findings of this study highlight the significant associations between diabetes mellitus (DM), knee osteoarthritis (KOA), and various health outcomes, including pain, stiffness, swelling, mobility, and health-related quality of life. Diabetes emerged as a strong determinant of KOA severity, consistent with prior work showing that metabolic abnormalities contribute to structural joint changes and symptom intensification. The current results align with Alazani et al., who reported greater severity of knee pain and bilateral involvement among diabetic patients with KOA, suggesting that insulin resistance and systemic inflammation accelerate cartilage degradation and worsen joint symptoms (36). Similarly, Courties and Sellam described how low-grade chronic inflammation and advanced glycation end products in diabetes may exacerbate synovial inflammation and cartilage loss, thereby explaining the more severe pain and stiffness reported by diabetic participants in this study (37).

The association between type 2 diabetes mellitus and higher KOA prevalence corroborates findings from Geng et al., who noted an increased incidence of KOA in older adults with T2DM, and from Goyal and Jiale, who observed that nearly one-third of diabetic patients developed osteoarthritis compared to the general population (38,39). These results suggest that beyond mechanical loading, metabolic dysfunction substantially contributes to KOA pathogenesis. In addition, the present study demonstrated significant relationships between diabetes and multiple dimensions of pain, including current, best, worst, and average pain ratings. This observation is consistent with Louati et al., who found that diabetes and KOA together lead to worse physical function and greater pain severity than either condition alone (40). Such synergy underscores the clinical importance of early glycemic control and integrated management strategies to mitigate the progression of KOA symptoms in diabetic populations.

Swelling, stiffness, and reliance on assistive devices were frequent among participants, further emphasizing the compounded disability when diabetes coexists with KOA. More than half of the cohort reported frequent joint swelling, particularly among diabetics, pointing to a role of systemic metabolic inflammation in accelerating joint degeneration. These findings parallel reports from Leite et al., who documented high rates of assistive device use among KOA patients with comorbid chronic conditions (41). The observed 39.1% device usage highlights the functional toll of this comorbidity and the need for tailored rehabilitation and mobility support programs.

Demographic trends also provide context for disease burden. The mean age of 48.9 years, with most participants between 40 and 60 years, is consistent with Vennu and Bindawas, who described increased KOA prevalence in individuals over 50, reflecting age as a critical determinant of cartilage degeneration (42). The nearly equal gender distribution differs from some prior research reporting greater symptom severity in women, such as Lee et al., who found higher pain and functional restriction among female patients with KOA (43). The present data suggest that, at least within this Faisalabad cohort, the burden of diabetes-related KOA is comparably distributed across genders, supporting generalizability of the findings to both sexes.

The quality-of-life analysis through the SF-36 revealed that diabetic participants had markedly lower scores in vitality, physical functioning, general health, and social and emotional roles, confirming the multidimensional impact of this comorbidity. These outcomes mirror Murillo et al., who reported poorer HRQoL in patients with chronic conditions like diabetes and osteoarthritis (44), and King et al., who demonstrated that comorbid OA and DM lead to disproportionate impairments in daily functioning (45). Interestingly, while most individual domains of the SF-36 showed significant associations with diabetes, the total score did not reach significance, possibly because aggregated scoring masked domain-specific deficits. This finding reinforces the importance of analyzing domain-level data to capture nuanced effects on patient well-being.

Lifestyle modifications such as dietary adjustment and exercise were adopted by 57.9% of participants, underscoring the role of non-pharmacologic strategies in managing these chronic conditions. This resonates with evidence from King and Rosenthal, who emphasized that lifestyle interventions improve pain, function, and quality of life in both OA and diabetes (46). However, the fact that nearly half of the participants had not implemented such measures reflects a gap in awareness and self-care, highlighting the need for patient education and culturally adapted intervention programs.

CONCLUSION

The strength of this study lies in its multidimensional approach, simultaneously assessing pain, physical impairment, lifestyle practices, and quality-of-life domains to provide a comprehensive understanding of the burden of KOA in diabetic versus non-diabetic populations. However, its cross-sectional design limits causal inference, and the relatively modest, single-city sample restricts generalizability. Moreover, the absence of data on glycemic control and body mass index prevents detailed exploration of potential confounders. Despite these limitations, the study adds important evidence from a South Asian setting where the dual burden of metabolic and musculoskeletal diseases is increasing but remains underexplored. Future research should employ longitudinal and multicenter designs with larger sample sizes to better establish causal pathways between diabetes and KOA progression, while adjusting for metabolic and lifestyle factors. Interventional trials testing integrated care models involving endocrinology, physiotherapy, and dietary counseling would also be valuable. Clinically, these findings highlight the importance of early detection, glycemic optimization, weight management, and structured rehabilitation in diabetic patients to reduce the risk and severity of KOA, ultimately improving functional independence and quality of life.

REFERENCES

- Alenazi AM, Alshehri MM, Al Othman S, Alqahtani BA, Rucker J, Sharma N, et al. The Association of Diabetes With Knee Pain Severity and Distribution in People With Knee Osteoarthritis Using Data From the Osteoarthritis Initiative. *Sci Rep*. 2020;10(1):3985.
- Al-Jarallah K, Shehab D, Abdella N, Al Mohamedi H, Abraham M. Knee Osteoarthritis and Associated Factors in Patients Attending Primary Health Care Clinics. *Kuwait Med J*. 2015;47(3):197–202.
- Alkan BM, Fidan F, Tosun A, Ardicoglu O. Quality of Life and Self-Reported Disability in Patients With Knee Osteoarthritis. *Mod Rheumatol*. 2014;24(1):166–71.
- Almanna SB, Koulis AB, Mohamed M, Alchemies NH, Sheena BB, Ibrahim ZM, et al. Home-Based Circuit Training Improves Blood Lipid Profile, Liver Function, Musculoskeletal Fitness, and Health-Related Quality of Life in Overweight/Obese Older Adult Patients With Knee Osteoarthritis and Type 2 Diabetes: A Randomized Controlled Trial During the COVID-19 Pandemic. *BMC Sports Sci Med Rehabil*. 2024;16(1):125.
- Courties A, Sellam J. Osteoarthritis and Type 2 Diabetes Mellitus: What Are the Links? *Diabetes Res Clin Pract*. 2016;122:198–206.
- Eitner A, Culvenor AG, Wirth W, Schaible HG, Eckstein F. Impact of Diabetes Mellitus on Knee Osteoarthritis Pain and Physical and Mental Status: Data From the Osteoarthritis Initiative. *Arthritis Care Res*. 2021;73(4):540–8.
- Geng R, Li J, Yu C, Zhang C, Chen F, Chen J, et al. Knee Osteoarthritis: Current Status and Research Progress in Treatment. *Exp Ther Med*. 2023;26(4):1–11.
- Goyal R, Jiale I. Diabetes Mellitus Type 2. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2018.
- Halim M, Halim A. The Effects of Inflammation, Aging and Oxidative Stress on the Pathogenesis of Diabetes Mellitus (Type 2 Diabetes). *Diabetes Metab Syndr*. 2019;13(2):1165–72.
- Hsu H, Siwiec RM. Knee Osteoarthritis. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2018.
- Kawano MM, Araújo ILA, Castro MC, Matos MA. Assessment of Quality of Life in Patients With Knee Osteoarthritis. *Acta Ortop Bras*. 2015;23(6):307–10.
- King K, Rosenthal A. The Adverse Effects of Diabetes on Osteoarthritis: Update on Clinical Evidence and Molecular Mechanisms. *Osteoarthritis Cartilage*. 2015;23:A25–6.
- King LK, Waugh E, MacKay C, Stanaitis I, Stretton J, Weisman A, et al. “It’s a Dance Between Managing Both”: A Qualitative Study Exploring Perspectives of Persons With Knee Osteoarthritis and Type 2 Diabetes Mellitus on the Impact of Osteoarthritis on Diabetes Management and Daily Life. *BMJ Open*. 2022;12(11):e061472.
- Lee Y, Lee SH, Lim SM, Baek SH, Ha IH. Mental Health and Quality of Life of Patients With Osteoarthritis Pain: The Sixth Korea National Health and Nutrition Examination Survey (2013–2015). *PLoS One*. 2020;15(11):e0242077.
- Leite AA, Costa AJG, Lima BdeAM, Padilha AVL, Albuquerque EC, Marques CDL. Comorbidities in Patients With Osteoarthritis: Frequency and Impact on Pain and Physical Function. *Rev Bras Reumatol*. 2011;51(2):118–23.
- Lin CP, Chung CH, Lu CH, Su SC, Kuo FC, Liu JS, et al. Glucagon-Like Peptide-1 Receptor Agonists Therapy to Attenuate the Risk of Knee Osteoarthritis and Total Knee Replacement in Type 2 Diabetes Mellitus: A Nationwide Population-Based Cohort Study. *Medicine*. 2025;104(6):e40231.
- Mora JC, Przkora R, Cruz-Almeida Y. Knee Osteoarthritis: Pathophysiology and Current Treatment Modalities. *J Pain Res*. 2018;11:2189–96.
- Nielen JT, Emans PJ, van den Bergh JP, Reijman M, Bierma-Zeinstra SM, Schram MT, et al. Association of Type 2 Diabetes Mellitus With Self-Reported Knee Pain and Clinical Knee Osteoarthritis: The Maastricht Study. *Diabetes Metab*. 2018;44(3):296–9.

19. Seow SR, Sumaiyah M, Teoh JJ, Yusup AM, Rajab NF, Ismail IS, et al. Combined Knee Osteoarthritis and Diabetes Is Associated With Reduced Muscle Strength, Physical Inactivity, and Poorer Quality of Life. *Sci Rep.* 2024;14(1):39986.
20. Yao M, Xu BP, Li ZJ, Zhu S, Tian ZR, Li DH, et al. A Comparison Between the Low Back Pain Scales for Patients With Lumbar Disc Herniation: Validity, Reliability, and Responsiveness. *Health Qual Life Outcomes.* 2020;18:166.