Review Article

Association of Metabolic Syndrome with Pain Severity and Physical Functioning in Patients with Knee Osteoarthritis

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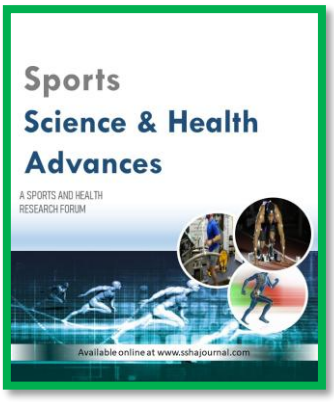

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Abstract

Background: Knee osteoarthritis (KOA) is a prevalent and debilitating musculoskeletal condition increasingly recognized as a metabolically influenced disorder. Metabolic syndrome (MetS)-encompassing obesity, hypertension, dyslipidaemia, and insulin resistance-has been implicated in the onset, progression, and symptomatic burden of KOA through both mechanical and systemic inflammatory mechanisms. **Objective**: This narrative review aimed to examine the association between metabolic syndrome and pain severity and physical functioning in patients with knee osteoarthritis. **Methods**: Studies published between 2001 and 2024 were reviewed, encompassing cross-sectional, longitudinal cohort and observational, case control studies. Key outcomes included pain severity, physical functioning, muscle strength, physical activity levels, and quality of life. **Results:** MetS was significantly more prevalent among KOA patients compared than in non-OA populations. A dose–response relationship was observed between MetS component accumulation and worse pain, functional decline, and disease progression. Elevated inflammatory markers, including IL-6, correlated with greater radiographic severity and disability. **Conclusion**: Metabolic syndrome significantly amplifies pain severity and functional impairment in KOA beyond mechanical loading alone. Integrated metabolic screening and multidisciplinary management are essential for optimizing patient outcomes.

Keywords: knee osteoarthritis, metabolic syndrome, pain severity, physical functioning, muscle strength, inflammation

Introduction

Knee osteoarthritis (KOA) is one of the most common musculoskeletal disorders in the world and is a major cause of chronic pain, physical disability, and reduced quality of life, especially in middle-aged and elderly people (Puenpatom et al.,2009; Coggon et al.,2001). The global prevalence of symptomatic KOA is increasing worldwide, in line with an ageing population and increasing rates of obesity, making it a significant public health concern (Sellam et al.,2013).

Once considered to be a simple degenerative disease arising primarily from mechanical joint damage, KOA is now recognized as a complex, multifactorial disease influenced by systemic, metabolic, and inflammatory processes (Yoshimura et al., 2012; Sellam et al.,2013).

This evolving understanding has led to a shift in clinical and research efforts to identify modifiable systemic risk factors that may contribute to the onset and progression of the disease beyond the biomechanical pathways (Clockaerts et al., 2010).

Metabolic syndrome (MetS), a complex set of metabolic disorders including central obesity, hypertension, dyslipidemia and insulin resistance or type 2 diabetes, has been identified as a major risk factor for KOA in epidemiological studies over the last few decades (Puenpatom et al.,2009; Wang et al., 2016) A large cross-sectional analysis of NHANES III data showed a significantly higher prevalence of MetS in knee OA compared to non-knee OA patients (Puenpatom et al.,2009).

The mechanisms by which MetS contributes to the development of KOA go beyond the mechanical loading of the joints in obese patients, which include the release of pro-inflammatory adipokines, like leptin and resistin, which, independently of the biomechanical load, induce chondrocyte degradation and inflammation (Clockaerts et al., 2010). Interleukin-6 (IL-6), C-reactive protein (CRP) and tumour necrosis factor-alpha (TNF-alpha) are implicated in the pathophysiology of OA (Samaan SF, Taha SI, 2022; Sellam et al.,2013).

While dyslipidemia may increase oxidative stress in joint tissues, hypertension has been shown to reduce subchondral bone blood flow and contribute to cartilage degeneration through microvascular mechanisms (Niu et al., 2017; Xie et al., 2017). Advanced glycation end-products (AGEs), which build up in cartilage and change its mechanical characteristics, making it more vulnerable to damage, are linked to diabetes and insulin resistance (Alenazi et al., 2020). These metabolic pathways, both separately and in concert, imply that MetS produces a systemic milieu that is favourable to the onset and exacerbation of Knee OA (Yoshimura et al., 2012; Wang et al., 2016).

MetS has been linked to more severe knee pain, increased functional disability, decreased muscle strength, and a lower quality of life in Knee OA patients (Tong et al., 2024; Seow et al., 2024; Li, H et al., 2016). The cumulative effect of metabolic burden on musculoskeletal health is highlighted by a dose-response relationship found between the quantity of MetS components present and the intensity of OA symptoms (Yoshimura et al., 2012). Additionally, patients with metabolic comorbidities and Knee OA have shown decreased response to traditional rehabilitation programs, suggesting the need for integrated, metabolically informed management strategies (Inoue, R et al., 2011).

Despite the growing evidence linking MetS to Knee OA, clinical management of Knee OA has historically been focused on analgesic therapy, weight reduction and surgical intervention, with little attention to the wider metabolic context (Sellam et al.,2013; Wang et al., 2016). There is an urgent need to synthesize the existing evidence on this association to inform more holistic and patient-oriented approaches to KOA management.

This narrative review summarizes the available evidence investigating the association of metabolic syndrome with pain and functional performance in patients with knee osteoarthritis and aims to highlight the clinical consequences of metabolic co-morbidity and support the development of integrated rehabilitation strategies.

Material and Methods

This review adopted a narrative design, drawing upon a literature base of peer-reviewed studies published between 2001 and 2024, each examining the relationship between metabolic syndrome and knee osteoarthritis. Study designs included cross-sectional investigations, prospective longitudinal cohort studies, case-control studies, observational analyses, providing a broad and complementary evidence base.

The main outcomes studied included metabolic syndrome and its individual components (obesity, diabetes, hypertension, dyslipidemia), as well as pain status as assessed by validated tools, including the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) and numeric pain scales (Tong et al., 2024; Samaan SF, Taha SI, 2022; Alenazi et al., 2020). Physical functioning was measured through performance-based assessments, including the Timed Up and Go (TUG) test, the Five-Times-Sit-to-Stand Test (FTSST), the Six-Minute Walk Test (6MWT), and handgrip dynamometry, as well as through patient-reported outcome measures, including the Short

Form-36 (SF-36) (Seow et al., 2024; Inoue, R et al., 2011). Physical activity levels, quality of life, muscle strength, and inflammatory markers, including IL-6 and CRP, were also examined across studies (Samaan SF, Taha SI, 2022; Clockaerts et al., 2010).

Table 1: Summary of Reviewed Studies

Author & Year	Study Design Participants (Sample Size)	Objective	Materials & Methods	Outcome Measures	Results
Tong et al., 2023	Cross-sectional; 178 symptomatic KOA patients ≥40 yrs.	To examine associations between body composition, physical activity, pain and function in KOA patients.	Body composition assessment; WOMAC-P, WOMAC-F; FTSST; regression analysis.	Pain; function; skeletal muscle index; bilateral pain.	Higher fat mass and lower physical activity associated with worse pain and function; greater muscle mass improved objective function.
Seow et al., 2023	Cross-sectional; 436 adults ≥50 yrs (261 with OA).	To investigate impact of combined knee OA and diabetes on muscle strength, physical performance, activity and quality of life.	Handgrip strength; TUG; 6MWT; 5xSTS; QoL questionnaires.	Muscle strength; physical performance; activity level; quality of life.	Combined OA and diabetes associated with reduced strength, poorer physical performance, inactivity and lower quality of life.
Samaan & Taha, 2022	Cross-sectional; 116 primary knee OA patients.	To evaluate association between metabolic syndrome and severity of primary knee OA.	MetS criteria; KL grading; HAQ-DI; WOMAC; IL-6 measurement.	Radiographic severity; disability; inflammatory marker.	MetS linked with more severe radiographic damage, higher disability and elevated IL-6 levels.
Inoue et al., 2011	Cross-sectional study; 795 Japanese adults.	To examine the association between metabolic syndrome and radiographic knee OA.	Radiographic assessment of knee OA and evaluation of metabolic syndrome components.	Knee OA, metabolic syndrome, hypertension, hyperlipidaemia, and diabetes.	Knee OA was associated with a higher prevalence of metabolic syndrome, particularly among women.
Alenazi et al., 2020	Cross-sectional (OAI, USA); N=1319.	To examine relationship between diabetes and knee pain severity in knee OA.	Diabetes status; numeric pain scale; multivariable regression.	Pain severity; pain distribution.	Diabetes independently associated with greater knee pain severity and bilateral involvement.
Niu et al., 2017	Prospective cohort (Framingham); N=991; ~10-year follow-up.	To determine longitudinal association between MetS and incident knee OA.	MetS (ATP III); radiographs; BMI-adjusted regression.	Incident radiographic & symptomatic OA.	MetS–OA association largely explained by BMI; high blood pressure remained significant.
Xie et al., 2017	Cross-sectional; Chinese adults	To assess association between MetS	MetS criteria; KL grading; logistic regression.	Radiographic OA;	MetS associated with OA prevalence, especially osteophyte formation;

	≥40 yrs (N=5764).	and radiographic knee OA features.		osteophytes; JSN.	risk increased with more components.
Li et al., 2016	Case-control study; 151 participants (70 knee OA patients, 81 controls).	To investigate the association between metabolic syndrome and knee OA symptom severity.	Assessment of metabolic syndrome components (obesity, hypertension, hyperglycaemia, dyslipidaemia) through clinical and laboratory evaluations.	VAS (pain), HSS Knee Score (knee function), HAMD (depression).	Metabolic syndrome and its components were associated with greater pain, depression, and poorer knee function in knee OA patients.
Shin, 2014	Cross-sectional (KNHANES, Korea); N=2363 ≥50 yrs.	To evaluate association between MetS, radiographic OA and knee pain.	MetS (NCEP-ATP III); KL grading; numeric pain scale; adjusted regression.	Radiographic OA; knee pain.	Association became non-significant after BMI adjustment; pain increased with more MetS components.
Yoshimura et al., 2012	Prospective cohort (ROAD, Japan); N=1690 baseline; 1384 follow-up.	To investigate effect of metabolic factors on incidence and progression of knee OA.	Metabolic assessment; knee radiographs at baseline and 3 years; KL grading.	Incidence & progression of knee OA.	Greater number of MetS components increased OA incidence and progression in dose–response manner.
Puenpatom & Victor, 2009	Cross-sectional (NHANES III, USA); N=7714 adults.	To determine association between MetS and osteoarthritis prevalence.	MetS (ATP III); OA clinically & radiographically diagnosed; logistic regression.	Prevalence of MetS & components.	MetS and its components significantly more prevalent in OA; strong association between OA and MetS.
Coggon et al., 2001	Case-control study; 675 participants (kneeOA cases and controls).	To investigate the relationship between obesity and knee osteoarthritis.	BMI measurement; occupational exposure questionnaire; radiographic assessment.	BMI, obesity status, and presence of knee osteoarthritis.	Obese individuals had significantly higher risk of knee OA; risk further amplified by physically demanding occupations, highlighting interaction between mechanical and metabolic factors.

Results

The findings from the reviewed studies consistently show that metabolic syndrome is significantly more common in patients with knee osteoarthritis than in patients without knee osteoarthritis (Puenpatom et al.,2009). An accumulation of MetS components was associated with a dose–response increase in OA risk and disease progression across multiple study designs (Yoshimura et al., 2012). While obesity plays a major mechanical role, the reviewed evidence indicates that metabolic abnormalities contribute to symptom severity through pathways extending beyond mechanical joint loading alone.

Pain Severity

A greater number of MetS components was consistently associated with higher knee pain severity, even after adjustment for body mass index (Shin D, 2014; Yoshimura et al., 2012). Hypertension, low high-density lipoprotein (HDL) cholesterol, and diabetes were each significantly linked with worse pain outcomes across multiple studies (Li, H. et al., 2016; Shin D, 2014). Diabetes emerged as an independent predictor of higher pain intensity and bilateral knee involvement in a large multivariate analysis (Alenazi et al.,

2020). MetS was also associated with structural features such as osteophyte formation, which may further contribute to the symptomatic burden experienced by affected patients (Xie et al., 2017).

Physical Functioning and Muscle Strength

Patients with combined KOA and metabolic disorders demonstrated markedly reduced muscle strength, including diminished handgrip strength, alongside poorer performance across objective functional assessments such as the TUG, 6MWT, and Five-Times-Sit-to-Stand Test (Seow et al., 2024). Increased physical inactivity and lower quality of life were additional characteristic findings in this population. Higher body fat mass was associated with worse pain and functional scores, while greater skeletal muscle mass was associated with improved objective functional performance (Tong et al., 2024).

Inflammatory and Functional Mechanisms

Elevated inflammatory markers, particularly IL-6, in MetS patients correlated with greater radiographic severity and functional disability (Samaan SF, Taha SI, 2022). Adipokines, including leptin and resistin, released from dysfunctional adipose tissue, were shown to promote chondrocyte degradation and synovial inflammation independently of mechanical loading (Clockaerts et al., 2010). Low-grade systemic inflammation, increased adiposity, and reduced muscle mass appear to collectively amplify pain and functional decline in this patient population (Sellam et al.,2013; Wang et al., 2016).

Discussion

The findings of the present review indicate that metabolic syndrome (MetS) is strongly associated with increased pain severity and reduced physical function in individuals with knee osteoarthritis (KOA). Several studies have demonstrated a significantly higher prevalence of MetS among patients with KOA compared to individuals without osteoarthritis, suggesting that metabolic abnormalities may play an important role in the development and progression of the disease beyond age-related joint degeneration alone (Puenpatom et al.,2009). The association appears to strengthen as the number of metabolic risk factors increases, with longitudinal evidence demonstrating a dose-dependent relationship between the accumulation of MetS components and the incidence and progression of KOA (Yoshimura et al., 2012).

Traditionally, obesity has been considered the principal link between MetS and KOA because of the increased mechanical loading imposed on weight-bearing joints. However, growing evidence suggests that metabolic abnormalities contribute to osteoarthritis through mechanisms that extend beyond biomechanical stress. A meta-analysis reported that MetS was associated with an increased risk of knee osteoarthritis, even after considering the contribution of obesity, indicating that metabolic disturbances themselves may influence disease pathogenesis. Furthermore, individual components of MetS, including diabetes mellitus, hypertension, and dyslipidemia, have been independently associated with greater pain severity and worse clinical outcomes in patients with KOA (Alenazi et al., 2020; Li, H. et al., 2016; Shin D, 2014).

One of the most important mechanisms underlying this association is chronic low-grade systemic inflammation. Metabolic syndrome is characterized by increased production of inflammatory mediators originating from adipose tissue, which contribute to cartilage degradation and synovial inflammation. Sellam and Berenbaum (2013) described osteoarthritis as a potential metabolic disease, highlighting the role of systemic inflammatory pathways in disease progression. Adipokines such as leptin, adiponectin, and resistin are produced by adipose tissue and can directly influence chondrocyte metabolism, extracellular matrix turnover, and inflammatory responses within the joint (Clockaerts et al., 2010). These mediators stimulate the production of matrix metalloproteinases and pro-inflammatory cytokines, accelerating cartilage breakdown and structural joint damage. Elevated circulating levels of inflammatory markers, particularly interleukin-6 (IL-6) and C-reactive protein (CRP), have also been associated with greater radiographic severity and functional impairment in patients with KOA (Samaan SF, Taha SI, 2022).

The reviewed studies further demonstrated that metabolic abnormalities adversely affect physical function and muscle performance. Individuals with both KOA and MetS consistently exhibited lower muscle strength, poorer performance on functional tests, and reduced levels of physical activity compared with individuals without metabolic dysfunction (Seow et al., 2024; Inoue et al., 2011). Higher body fat mass was associated with worse pain and disability scores, whereas greater skeletal muscle mass was associated with superior functional performance (Tong et al., 2024). These findings suggest that the interaction between adiposity, sarcopenia, and metabolic dysfunction contributes substantially to functional decline. Reduced muscle strength may compromise joint stability and movement efficiency, thereby increase pain and disability while accelerate disease progression.

The concept of a "metabolic osteoarthritis phenotype" has emerged in recent years to describe a subgroup of patients in whom metabolic and inflammatory factors play a dominant role in disease expression (Sellam et al.,2013). Unlike the traditional mechanical model of osteoarthritis, this phenotype emphasizes the contribution of systemic metabolic disturbances, chronic inflammation, adipose tissue dysfunction, and altered body composition. Recognition of this phenotype has important clinical implications because it suggests that management strategies focusing exclusively on joint symptoms may be insufficient for many patients with KOA and concurrent MetS.

Longitudinal studies provide additional support for a causal relationship between metabolic dysfunction and osteoarthritis progression. In the Framingham Osteoarthritis Study (2022), MetS was associated with an increased risk of incident symptomatic and radiographic KOA over a ten-year period, although adjustment for body mass index attenuated the strength of the association, highlighting the important contribution of obesity. Similarly, the ROAD study by Yoshimura et al., (2012) demonstrated that increasing numbers of metabolic risk factors were associated with a stepwise increase in both the occurrence and progression of knee osteoarthritis. Together, these findings suggest that metabolic abnormalities may influence not only symptom severity but also structural disease progression.

From a rehabilitation perspective, the findings underscore the importance of addressing metabolic health alongside conventional physiotherapy interventions. Exercise therapy remains a cornerstone of KOA management and offers benefits that extend beyond improvements in muscle strength and joint function. Regular physical activity has been shown to improve insulin sensitivity, reduce systemic inflammation, enhance cardiovascular health, and favourably modify body composition. Therefore, integrating aerobic exercise, resistance training, weight-management strategies, and metabolic risk-factor control into rehabilitation programmes may provide superior outcomes compared with symptom-focused treatment alone.

Despite the consistency of the available evidence, several limitations should be acknowledged. Many studies included in the review were cross-sectional in nature, limiting the ability to establish causality. Considerable heterogeneity existed in the diagnostic criteria used for MetS, participant characteristics, and outcome measures, making direct comparisons between studies challenging. Additionally, because obesity frequently coexists with other metabolic abnormalities, it remains difficult to fully distinguish the independent effects of individual MetS components on KOA outcomes (Niu et al., 2017; Shin D, 2014). Future prospective studies with standardized diagnostic criteria and outcome measures are needed to further clarify the mechanisms linking metabolic dysfunction and osteoarthritis and to evaluate targeted interventions for patients with the metabolic OA phenotype.

Overall, the available evidence supports the view that knee osteoarthritis is not solely a degenerative joint disorder but also a condition influenced by systemic metabolic dysfunction. Recognition of the metabolic contribution to KOA may facilitate earlier identification of high-risk individuals and promote the development of more comprehensive, multidisciplinary treatment approaches aimed at improving both metabolic health and musculoskeletal outcomes.

Conclusion

The available evidence indicates that metabolic syndrome is associated with greater pain severity, reduced muscle strength, impaired physical performance, and poorer quality of life in individuals with knee osteoarthritis. A dose-response relationship suggests that the accumulation of metabolic abnormalities progressively worsens clinical outcomes. Although obesity remains an important mechanical contributor, diabetes, hypertension, dyslipidemia, and systemic inflammation also appear to influence disease severity independently.

These findings support the view that knee osteoarthritis should not be considered solely a degenerative joint disorder but also a condition influenced by metabolic dysfunction. Routine screening for metabolic syndrome components and the integration of metabolic management with rehabilitation programmes may improve patient outcomes. A multidisciplinary approach that combines physiotherapy, lifestyle modification, and metabolic risk-factor control is likely to provide the greatest benefit for individuals with knee osteoarthritis.

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