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Exploring Associations: Lipid Profiles, Body Composition, Anthropometry, and Osteoarthritis Index

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ABSTRACT

Introduction. Osteoarthritis (OA) is a joint disorder and is considered one of the leading causes of disability worldwide. It is commonly associated with aging and characterized by chronic pain, joint stiffness, and limitations in daily activities. This study aimed to evaluate the association between lipid profile and body composition with the severity of OA, as measured by the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) score. **Methods.** This cross-sectional study involved 67 community-dwelling older adults. Assessments included fasting lipid profiles (HDL, LDL, total cholesterol, Apo A, Apo B, and triglycerides), body fat distribution and muscle mass, anthropometric measurements (body mass index, waist circumference, and hip circumference), and handgrip strength. Associations between variables were analyzed using Spearman's correlation test with a significance level of 5%. **Results.** Correlation analysis demonstrated associations between lipid profile levels and worsening OA symptoms. Higher Apo B levels were associated with increased trunk fat ($r=0.47$, $p<0.001$) and greater joint pain ($r=0.35$, $p=0.004$). Lower HDL levels were associated with increased visceral fat ($r=-0.37$, $p=0.002$) and greater joint stiffness ($r=0.26$, $p=0.038$). Greater upper extremity muscle mass was associated with less severe pain ($r=-0.38$, $p<0.001$), while higher body mass index and waist circumference were associated with higher WOMAC scores ($r=0.32$, $p=0.009$). **Conclusion.** These findings support the concept that OA is a systemic condition influenced by metabolic and biomechanical pathways. OA management should therefore include metabolic control, optimization of body composition, and routine monitoring of lipid profiles and anthropometric indices as part of non-pharmacological therapy and preventive strategies.

1. Introduction

Osteoarthritis (OA), particularly of the knee joint, is a leading cause of musculoskeletal disability worldwide. This condition is characterized by chronic pain, joint stiffness, and significant functional limitations in daily activities, especially among the older population.[1] According to estimates from the Global Burden of Disease (GBD) 2019 study, OA contributes substantially to global Years Lived with Disability (YLD).[2] A meta-analysis involving Asian populations reported a relatively high burden of knee osteoarthritis, with prevalence exceeding 5.5%, corresponding to approximately 5,677 cases per 100,000 individuals in Southeast Asia.[2,3] In Indonesia, epidemiological data indicate that the prevalence of osteoarthritis more than doubled between 1990 and 2019, underscoring the significant public health implications of OA.[4] Data from urban areas such as Jakarta reveal a high prevalence of general and central obesity accompanied by rapid lifestyle transitions, positioning anthropometric parameters as critical determinants of musculoskeletal health in the community. These parameters are directly relevant to the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), which assesses pain, stiffness, and physical function in individuals with osteoarthritis.[5,6]

Anthropometric factors, such as body mass index (BMI) and waist circumference, strongly influence the clinical manifestations of OA. Several previous studies have demonstrated a dose–response relationship between higher BMI and worse WOMAC scores in the pain and physical function domains, reinforcing obesity as a major determinant of OA clinical severity.[7] Increased waist circumference, as an indicator of central adiposity, has been associated with higher WOMAC scores and reduced quality of life among OA patients, suggesting that fat distribution

may be more clinically relevant than overall body mass alone.[8,9] In addition to BMI and waist circumference, handgrip strength has shown a negative correlation with WOMAC scores, reflecting reduced muscle strength and functional capacity in patients with OA.[10,11]

Beyond simple anthropometric measurements, body composition offers deeper biological insight into WOMAC domains. Higher fat mass has been correlated with more severe pain and functional impairment, reflecting both mechanical and metabolic contributions of adiposity to OA symptoms.[12] The phenomenon of sarcopenic obesity, characterized by increased fat mass combined with reduced muscle mass, has been associated with worse clinical outcomes in OA. Therefore, combined assessments of skeletal muscle mass percentage and body fat percentage are essential to enrich functional analyses and provide a more comprehensive understanding of disease impact.[13,14] In Jakarta and other major urban centers in Indonesia, low physical activity levels, coupled with a high prevalence of central obesity, further underscore the urgency of incorporating body composition variables to explain variations in pain, stiffness, and functional outcomes.[15]

Growing clinical evidence indicates that abnormal lipid profiles (dyslipidemia) are also associated with OA symptom progression, with hypercholesterolemia being linked to greater knee pain intensity in OA patients. These associations highlight the role of inflammatory pathways and metabolic dysfunction in OA pathogenesis.[16] Recent evidence suggests that dyslipidemia, characterized by elevated total cholesterol and triglycerides and reduced high-density lipoprotein (HDL) levels, contributes to structural knee damage and the development of a metabolic osteoarthritis phenotype. Furthermore, circulating apolipoprotein B levels have been consistently reported to be higher in individuals with osteoarthritis compared to those without the disease. These findings underscore the importance of measuring apolipoprotein A1 and apolipoprotein B in studies exploring the metabolic mechanisms underlying osteoarthritis.[17,18] The high prevalence of metabolic syndrome components, particularly low HDL levels and central obesity, further reinforces the importance of including total cholesterol, HDL, triglycerides, apolipoprotein A1, and apolipoprotein B as meaningful independent variables in the context of OA.[19]

Overall, the increasing burden of osteoarthritis and the significant growth rate of Years Lived with Disability (YLD) emphasize the urgency of research aimed at mapping the biological pathways linking WOMAC symptom domains with dominant metabolic and morphometric risk factors in urban populations.[1,2] Previous studies have indicated that data elucidating the combined relationships between anthropometric parameters, body composition, lipid profiles, and OA symptoms remain limited.[20] Therefore, this study aims to map the interrelationships and potential pathways linking lipid profiles, body composition, and anthropometric parameters with WOMAC scores in an adult population in Jakarta, thereby providing a foundation for preventive interventions targeting metabolic and musculoskeletal risks within urban primary healthcare settings.

2. Methods

This is a cross-sectional study that recruited 65 community-dwelling older adults aged 60 years or older from the Asisi Church community in Tebet, South Jakarta, using consecutive sampling at community gatherings and health outreach events within a defined enrollment window. The inclusion criteria were age >60 years, ability to provide informed consent, ability to ambulate independently or with minimal aid, and ability to complete the WOMAC assessment. For the exclusion criteria, any diagnosed inflammatory arthritis (e.g., rheumatoid, psoriatic, gout), prior major lower-limb joint replacement, acute knee trauma or surgery within the last 3 months, decompensated acute illness, severe cognitive impairment limiting reliable assessment, presence of implanted electronic cardiac devices (contraindication for bioimpedance), or conditions that may substantially distort bioimpedance measures (e.g., gross edema). The final sample size (n=65) was pragmatic and community-based; with alpha 0.05 and power 80%, this sample provides adequate precision to detect at least moderate correlations (approximately $|\rho| \geq 0.33$), appropriate for the primary objective of correlation mapping rather than causal inference.

Data collection followed standardised operating procedures by trained assessors under the supervision of a study clinician. Lipid profiles were obtained as follows: LDL, HDL, total cholesterol, and triglycerides were measured using a capillary-based lipid panel (Nesco) according to the manufacturer's protocol, while apolipoproteins A1 (ApoA1) and apolipoproteins B (ApoB) were quantified from venous serum using a semi-automated chemical analyser under internal quality control. Body composition, including total body fat percentage, fat distribution indices, and skeletal muscle percentage, was measured using bioelectrical impedance analysis (Karada OMRON HBF 375) with participants barefoot, following pre-test recommendations (voiding, removal of metal accessories, and short rest). Height was measured to 0.1 cm using a Microtoise GEA stadiometer with Frankfurt plane alignment. Body weight was captured using an OMRON device and used to compute BMI. Waist circumference was measured at the midpoint between the lower costal margin and the iliac crest, and hip circumference at the maximal gluteal

prominence, both using a non-stretch elastic tape to 0.1 cm, with two readings averaged (third reading if discrepancy >0.5 cm). Handgrip strength was measured using a Camry EH 101 dynamometer in the seated position with the shoulder adducted, the elbow at 90°, and the neutral wrist. Three trials were performed on the dominant hand with 30 - 60 seconds of rest, and the highest value (kg) was recorded. Symptom burden was assessed using the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) administered as a structured questionnaire.

Independent variables comprised lipid profiles (LDL, HDL, total cholesterol, triglycerides, ApoA1, ApoB), body composition (total body fat percentage, skeletal muscle percentage, and fat distribution metrics from OMRON HBF 375), anthropometric parameters (BMI in kg/m^2, waist circumference in cm, hip circumference in cm), and handgrip strength (kg). The dependent variable was the Osteoarthritis Index, operationalized by the WOMAC scores, including the total score and the three subscales: pain, stiffness, and physical function. Operational definitions were as follows BMI was calculated as weight (kg)/height^2 (m^2), waist and hip circumferences were the averaged centimeter measures as per protocol, handgrip strength was the best of three dominant-hand trials in kilograms, lipid parameters were expressed in mg/dL per device or analyzer calibration, ApoA1 and ApoB were reported in mg/dL, and body composition outputs were expressed as percentages provided by the OMRON device (total body fat %, skeletal muscle %, and region-specific fat distribution where applicable). WOMAC responses were summed by domain and either analyzed as raw sums or transformed to a 0–100 scale (higher scores indicating worse symptoms), with the chosen scaling applied consistently across analyses and reported in the statistical plan. If any data are missing, they will be removed.

Statistical analysis adhered to a prespecified plan. Univariate analysis summarized continuous variables by median (depending on distribution) and categorical variables as counts (percentages). Distributional assumptions were evaluated using the Kolmogorov–Smirnov test and visual inspection (histograms). Given the anticipated non-normality for several clinical variables, bivariate associations between each independent variable and WOMAC outcomes (total, pain, stiffness, function) were tested using Spearman's rank correlation (r) with a two-tailed alpha of 0.05. Where appropriate, sensitivity analyses explored robustness (e.g., by excluding a priori-identified outliers), while acknowledging the exploratory nature of multiple comparisons without formal multiplicity adjustment. All analyses targeted 80% power at alpha 5% to detect moderate effect sizes within the constraints of n=65, and were performed using SPSS, with complete-case analysis after documenting patterns of missingness and applying double data entry and range checks for quality control.

3. Results

The study included 65 respondents who met the inclusion criteria, with a median age of 74 years (range 62–88) and a predominance of females (72.3%). The assessment of osteoarthritis symptoms using the WOMAC index showed relatively low median scores for pain (2), stiffness (1), and physical function (4), with a median total WOMAC score of 9, though wide ranges indicated variability in disease severity. Body composition analysis revealed a median total skeletal muscle percentage of 22.8%, with the trunk (16.6%), the upper extremities (26.8%), and the lower extremities contributing the largest proportions (37.2%). While total body fat reached a median of 34.7% and visceral fat a median of 10. Anthropometric parameters showed a median BMI of 24.1 kg/m^2, a waist circumference of 88.5 cm, a hip circumference of 96 cm, and a waist-to-hip ratio of 0.921, reflecting a generally overweight profile. Laboratory measurements showed a median total cholesterol of 205 mg/dL, HDL of 51 mg/dL, triglycerides of 132 mg/dL, LDL of 129 mg/dL, ApoA of 160 mg/dL, and ApoB of 105 mg/dL. In terms of physical function, the median handgrip strength was 18.4 kg, suggesting reduced muscular performance consistent with older adult populations (Table 1).

The correlation analysis showed that WOMAC scores (total, pain, stiffness, and physical function) were strongly interrelated ($r = 0.39$-0.97, $p < 0.05$), confirming the internal consistency of these symptom domains. Skeletal muscle parameters demonstrated negative correlations with WOMAC outcomes ($r = -0.26$ to -0.38, $p < 0.05$), suggesting that higher muscle mass may protect against greater symptom severity. In contrast, body fat distribution, particularly visceral and subcutaneous fat, showed positive associations with WOMAC scores ($r = 0.26$-0.35, $p < 0.05$), suggesting that greater adiposity is significantly associated with worse osteoarthritis symptoms. Within body composition, skeletal muscle and fat compartments showed strong inverse correlations ($r = -0.67$ to -0.93, $p < 0.001$), whereas fat compartments themselves were highly intercorrelated ($r = 0.79$ to 0.99, $p < 0.001$) (Figure 1-4).

Table 1. Characteristics of respondents

Parameter	Median (%)	Minimum	Maximum
Age, years	74	62	88
Gender (%)			
Male	18 (27.7)		
Female	47 (72.3)		
Pain (WOMAC)	2	0	13
Stiffness (WOMAC)	1	0	6
Physical Function (WOMAC)	4	0	52
Total WOMAC Score	9	0	69
Total Skeletal Muscle %	22.8	17.6	36
Skeletal Muscle, Trunk %	16.6	11.8	27.8
Skeletal Muscle, Upper Ext %	26.8	15.3	37.1
Skeletal Muscle, Lower Ext %	37.2	24.4	59.5
Total Body Fat %	34.7	10.6	43.8
Visceral Fat	10	0.5	70
Body Mass Index, kg/m^2	24.1	15.6	35.6
Total Subcutaneous Fat %	27.1	10.7	70.4
Subcutaneous Fat, Trunk %	23.7	9	34.1
Subcutaneous Fat, Upper Ext %	40.6	13.9	56.6
Subcutaneous Fat, Lower Ext %	34.2	13.3	47.8
Total Cholesterol (mg/dL)	205	135	332
High-Density Lipoprotein (HDL, mg/dL)	51	24	95
Triglycerides (mg/dL)	132	46	554
Low-Density Lipoprotein (LDL, mg/dL)	129	17	174
Apolipoprotein A1 (mg/dL)	160	96	177
Apolipoprotein B (mg/dL)	105	65	133
Weight (kg)	56.8	39.8	96.9
Height (cm)	154.8	139.5	174
Waist Circumference (cm)	88.5	66	114
Hip Circumference (cm)	96	78	126
Waist-to-Hip Ratio	0.921	0.777	1.030
Handgrip Strength (kg)	18.4	8.15	34.5

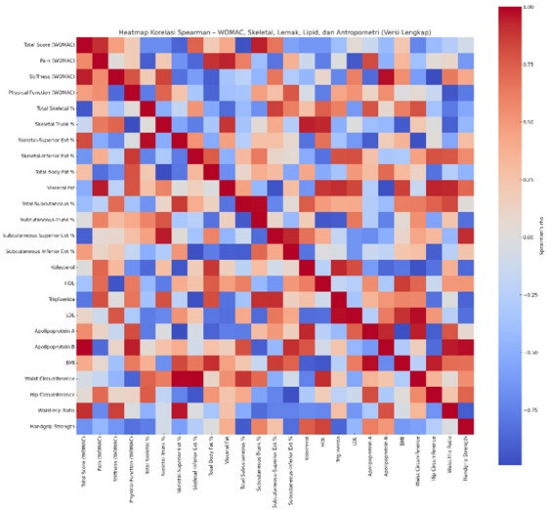

Figure 1. Correlation heatmap of WOMAC, skeletal muscle, body fat, lipid profile, and anthropometric parameters

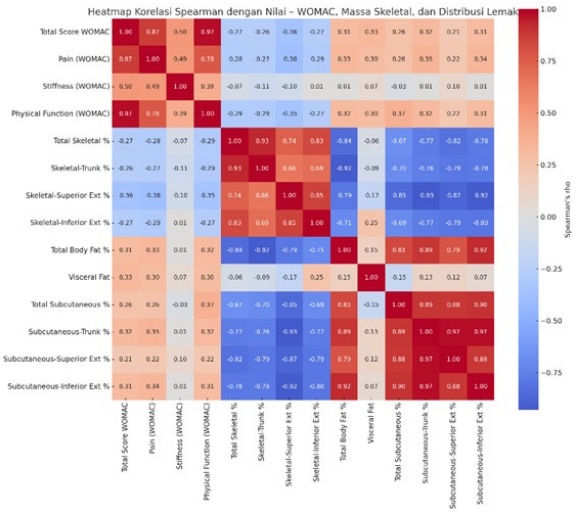

Figure 2. Correlation heatmap of WOMAC, skeletal muscle, and lipid distribution

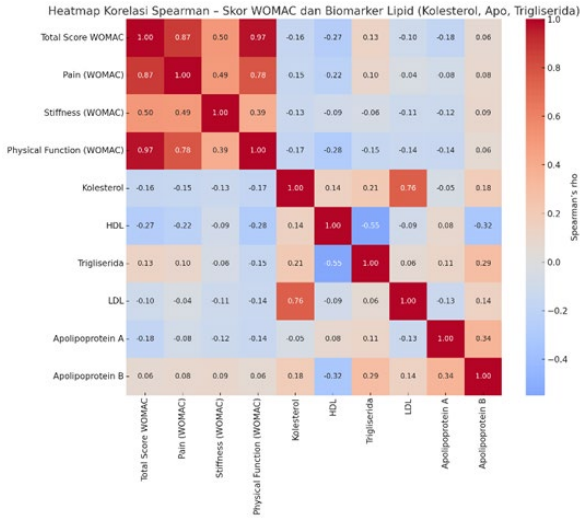

Figure 3. Correlation heatmap of WOMAC and lipid profile

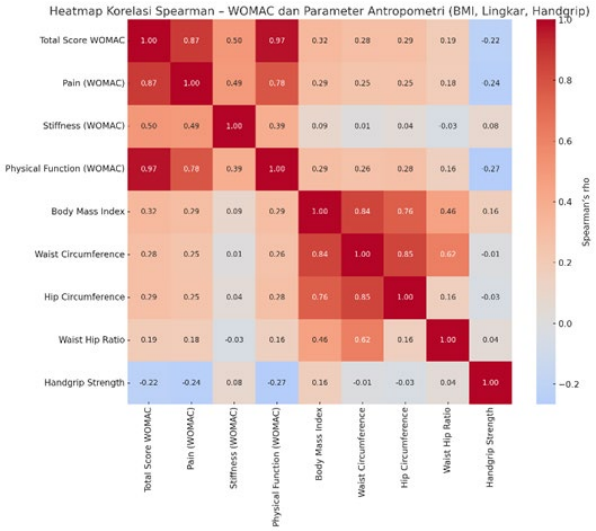

Figure 4. Correlation heatmap of WOMAC and anthropometric

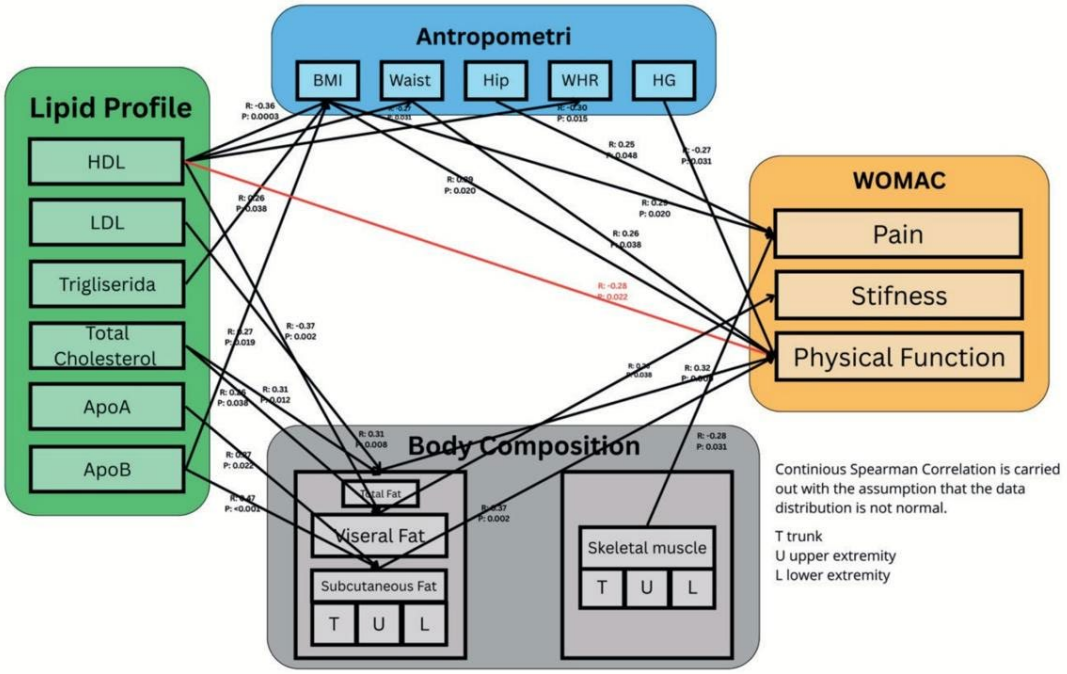

Figure 5. Correlation pathway diagram linking lipid profile, anthropometric measures, body composition, and WOMAC domain

For biochemical and anthropometric measures, WOMAC scores showed weak negative correlations with total cholesterol, HDL, LDL, and Apo-A (r = -0.04 to -0.28, p > 0.05), suggesting no significant associations. Conversely, triglycerides and Apo-B showed weak positive correlations with the WOMAC domains (r = 0.06-0.13, p > 0.05), though these were not statistically significant. Anthropometric indices such as BMI, waist circumference, and hip circumference showed modest positive correlations with WOMAC scores (r = 0.25-0.32, p < 0.05), suggesting that central obesity contributes to symptom burden. Notably, handgrip strength showed weak negative correlations with WOMAC outcomes (r = -0.22 to -0.27, p < 0.05), suggesting that better muscle function may mitigate osteoarthritis-related disability. Taken together, the results emphasize that significant predictors of WOMAC burden are primarily related to adiposity and muscle strength, whereas lipid alterations show only marginal, nonsignificant effects (Figure 1-4).

The correlation diagram illustrates the interrelationships among lipid profile, anthropometry, body composition, and WOMAC domains. Significant negative correlations were observed between skeletal muscle mass and WOMAC physical function (r = -0.28, *p* = 0.031), indicating that greater muscle mass is associated with better functional status, while visceral and subcutaneous fat showed positive correlations with WOMAC scores (r

= 0.31 to 0.32, $p < 0.05$), suggesting that higher adiposity contributes to worse osteoarthritis symptoms. Anthropometric indices such as BMI, waist, and hip circumference correlated positively with fat distribution (r = 0.25 to 0.37, $p < 0.05$), reflecting their role as markers of central obesity. In the lipid profile, HDL was inversely correlated with BMI (r = -0.36, $p = 0.0003$) and physical function (r = -0.28, $p = 0.022$), while triglycerides, total cholesterol, and ApoB demonstrated positive correlations with adiposity measures and WOMAC outcomes (r = 0.26 to 0.32, $p < 0.05$). (Figure 5)

4. Discussion

The findings of this study demonstrate patterns consistent with the international literature. WOMAC scores (pain, stiffness, and physical function) showed negative correlations with skeletal muscle mass parameters (r = -0.26 to -0.38) and positive correlations with adiposity indicators, including visceral and subcutaneous fat (r = 0.26 to 0.35). In contrast, the associations between WOMAC scores and lipid biomarkers were relatively weak, with triglycerides and apolipoprotein B showing modest positive correlations (r = 0.06-0.13), while HDL cholesterol and total cholesterol tended to show negative correlations (r = -0.04 to -0.28). The relationship between obesity, osteoarthritis risk, and disease severity has been extensively documented in prior research. Meta-analytic evidence indicates that overweight status increases the risk of knee pain (pooled OR 1.98), while obesity more than doubles the risk (pooled OR 2.66). Our findings align with this evidence, as positive correlations were observed between BMI, waist circumference, and WOMAC scores in our sample (r = 0.25-0.32). Collectively, these results support the concept of mechanical loading as a principal pathway linking adiposity to osteoarthritis symptomatology.[21,22]

Beyond mechanical loading, central fat distribution, as reflected by waist circumference and abdominal obesity, also demonstrates a strong correlation with functional limitations in osteoarthritis. Cohort studies and observational analyses have consistently reported that both general and abdominal obesity increase the risk of developing osteoarthritis. Available evidence indicates an odds ratio of 1.73 for several definitions of abdominal obesity. These findings corroborate the observed correlations between waist-to-hip ratio and WOMAC scores in the present study. Taken together, the results reinforce the role of central adiposity in contributing not only to localized mechanical stress but also to a pro-inflammatory metabolic phenotype associated with osteoarthritis progression.[23,24]

The role of muscle mass and functional strength, as measured by handgrip, appeared to be protective in our study. Handgrip strength demonstrated negative correlations with WOMAC scores (r = -0.22 to -0.27). Several studies and metal analysis have reported that sarcopenia or low muscle mass increases the odds of osteoarthritis and its related symptoms. Prior evidence has indicated that sarcopenia is associated with an odds ratio of 2.29, while low muscle mass index in both extremities confers an odds ratio of 1.36. Longitudinal studies have further demonstrated associations between lower extremity strength, handgrip performance, and the progression of pain and functional decline in osteoarthritis. Collectively, these findings suggest that loss of muscle mass and declining strength contribute to an increased functional burden and exacerbate the joint's susceptibility to mechanical stress.[25,26,27]

Based on the findings of our study, lipid profile data demonstrated relatively weak associations between classical lipid biomarkers (total cholesterol, LDL, and HDL) and WOMAC scores, although stronger positive correlations were observed for triglycerides and apolipoprotein B. Previous evidence has reported heterogeneous results: some studies identified associations between triglycerides or insulin resistance indices and the occurrence of osteoarthritis or subchondral lesions, whereas systematic reviews on dyslipidemia and osteoarthritis have documented variable outcomes depending on study design and population characteristics. Causal and experimental analyses have further suggested that LDL and triglycerides may promote synovial inflammation and joint tissue lesions in animal models. Accordingly, the weak associations observed in our cohort may reflect more subtle metabolic effects on osteoarthritis symptomatology, rather than the more pronounced mechanical influence of obesity.[28,29]

Overall, the correlation patterns observed in our sample support a multifactorial model of osteoarthritis pathogenesis. The findings suggest that obesity, particularly central fat distribution, together with reduced muscle mass and strength, plays a critical role in the development of osteoarthritis symptoms. These factors exert their influence through both mechanical loading and inflammatory–metabolic pathways. In contrast, lipid profiles showed more complex, relatively weaker direct associations with clinical outcomes. Furthermore, the strong intercorrelations among the WOMAC domains (r = 0.87-0.97) observed in this study highlight the clinical cohesion of pain, stiffness, and functional impairment as interrelated manifestations of the disease. This study has several limitations that warrant consideration in future research. The cross-sectional design limits the ability to draw causal inferences about the relationships among adiposity, muscle mass, lipid profiles, and osteoarthritis symptoms. As such, the observed associations may reflect bidirectional relationships, for example, osteoarthritis-related pain leading to reduced physical activity and subsequent muscle loss. In addition, the relatively weak associations of lipid

biomarkers in the correlational analyses may have been influenced by variability in lipid-lowering pharmacotherapy or residual metabolic confounding factors that were not fully controlled. The sample size and age distribution of participants may also limit the generalizability of the findings to older osteoarthritis populations or patients with more advanced radiographic disease. To address these gaps, prospective longitudinal studies incorporating repeated measures of body composition and metabolic and inflammatory biomarkers, such as IL-6, CRP, and adipokines, together with strict control for medication use and lifestyle factors, are recommended. Furthermore, interventional studies, including programs to enhance muscle mass or reduce central adiposity, are needed to determine whether modification of these targets can reduce WOMAC scores and slow radiographic progression of osteoarthritis.

5. Conclusion

This study concludes that osteoarthritis symptoms, as measured by WOMAC scores among older adults living in urban communities in Jakarta, are influenced by a complex interaction between anthropometric factors, body composition, muscle strength, and lipid profiles, in line with the study objective of mapping the relationships between morphometric and metabolic factors and the burden of OA symptoms. Anthropometric parameters reflecting general and central obesity, including body mass index, waist circumference, and hip circumference, were associated with higher WOMAC scores, suggesting a role for mechanical loading in joint pain, stiffness, and functional limitation. In addition, body composition provided further biological insight, with increased body fat and unfavorable fat distribution associated with symptom worsening, while greater muscle mass and higher handgrip strength demonstrated protective associations with physical function. Lipid profiles showed weaker but consistent associations, particularly for triglycerides and apolipoprotein B, suggesting the involvement of metabolic pathways in the pathogenesis of osteoarthritis. Overall, these findings support the concept of osteoarthritis as a systemic condition influenced by both mechanical and metabolic factors and underscore the importance of integrated management and prevention strategies through weight control, optimization of body composition and muscle strength, and monitoring of metabolic factors within urban primary healthcare settings.

6. Author Contribution

D.D, M.F.D, and S.G.B. contributed to literature review, data entry, and manuscript drafting. S.G. conceptualized the study, supervised data collection, and led the interpretation of results. A.H.S. assisted with methodological development and data interpretation. Y.F. contributed to study design, statistical analysis, and manuscript editing. All authors reviewed and approved the final version of the manuscript.

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