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[1]Faculty of Medicine, University of Jember, East Java, Indonesia
[2]Laboratory of Anatomy and Surgery, Faculty of Medicine, University of Jember, East Java, Indonesia
[3]Department of Otorhinolaryngology-Head and Neck Surgery, Dr. Soebandi Regional Hospital-Faculty of Medicine, University of Jember, East Java, Indonesia
[4]Department of Internal Medicine, Dr. Soebandi Regional Hospital-Faculty of Medicine, University of Jember, East Java, Indonesia
[5]Laboratory of Public Health and Community Medicine, Faculty of Medicine, University of Jember, East Java, Indonesia

*Correspondence to: ulfa.fk@unej.ac.id

Association between body shape index and body mass index with knee osteoarthritis: a case control study

Anik Nurmalikah,[1] Ulfa Elfiah,[2*] Nindya Shinta Rumastika,[3] Yuli Hermansyah,[4] Dwita Aryadina Rachmawati[5]

ABSTRACT

Background and purpose: Being overweight or obese, which can be determined using the body mass index (BMI), is one of the risk factors for knee osteoarthritis (KOA). Whilst, body shape index (BSI) is considered as more accurate indicator, hence, this study aims to assess correlation of BSI and BMI with KOA.

Methods: This study employed a case control design conducted in Jenggawah Village, East Java. Samples were randomly taken from the social service program of the Faculty of Medicine, University of Jember which conducted on June 18, 2023, included 30 KOA patients and 30 non-KOA patients who met the inclusion and exclusion criteria. The instruments used were Omni Calculator and KOA clinical criteria sheet. The statistical analysis used the Spearman Rank correlation test with a significance level of $p<0.05$.

Results: The statistical analysis revealed a significant, moderate, and direct correlation between BSI and KOA ($p=0.002$; $r=0.391$). On the other hand, BMI showed an insignificant, very weak, and inverse correlation with KOA ($p=0.432$; $r=-0.103$). BSI values indicate low amount of skeletal muscle mass with increased visceral or abdominal adiposity. BMI only assesses overweight, while BSI can represent central adiposity, which also affects KOA.

Conclusion: The study concludes that BSI is related to the incidence of KOA, while BMI is not related to the incidence of KOA in Jenggawah Village, Jember Regency. Future research could use a cohort plan to validate whether BMI and BSI can predict the incidence of KOA.

Keywords: knee osteoarthritis, body mass index, body shape index, obesity

INTRODUCTION

Osteoarthritis (OA) is a degenerative joint disorder that is characterized by pathological changes, including articular cartilage damage, subchondral sclerosis, osteophytes, synovial membrane inflammation, and paresis of the muscles around the joint, which can restrict movement.[1,2] Between 1990 and 2019, the number of people with OA worldwide increased by 113%, reaching a total of 528 million.[3] In Indonesia, OA was the most common rheumatic condition in 2010 and 2007, with 87% were knee osteoarthritis (KOA) and affects 12.7% of women and 15.5% of men radiologically between the ages of 40-60 years.[1,4]

KOA is triggered by several risk factors, including older age, female gender, obesity, heavy physical activity, muscle weakness, knee misalignment, and previous trauma.[4] The prevalence and severity of OA increase with age. OA is uncommon in individuals under 40 years old but prevalent in those over 60 years old.[1] The incidence of OA is nearly equal between men and women who are 50 years old or post-menopausal, caused by hormone.[5]

The risk factors for physical activity depend on the type and intensity of the activity. Endurance activities are less risky than strength activities.[6] Physical activity involves repetitive joint movements that can increase the risk of OA by putting pressure and load on the joints. KOA patients often limit knee movement to avoid pain, leading to atrophy and weakness in the quadriceps muscles, the largest muscle in the knee joint and plays a crucial role in absorbing energy and pressure.[7]

Obesity is a risk factor for OA as it causes an uneven distribution of mechanical loads on the knee joint and increases the burden on the knee joint, which is one of the body's supports. Furthermore, it alters behavioral factors, such as decreased physical activity, which reduces the ability and protective strength of the muscles surrounding the joint.[7–9] Infrapatellar fat pad, a fatty tissue that can produce adipokines: *adiponectin, leptin*, *visfatin*, and *resistin*, can be found on the back of the patella in obese individuals. Dysregulation of adipokines can lead to the secretion of pro-inflammatory factors.[7]

Articular cartilage lacks the capacity to regenerate, rendering it susceptible to lesions that can activate inflammatory pathways, degrading enzymes and the extracellular matrix (ECM) in articular cartilage.[10,11] Secretion of cytokines IL-8 and TNF-α by chondrocytes and synovial cells decrease synthesis of type 2 collagen and proteoglycans (which produce cartilage matrix) and increase catabolic and inflammatory mediators (as matrix-breaking enzymes) such as metalloproteases, cytokines, prostaglandin E2, leukotrienes, growth factors (TGF-β, FGF, VEGF, NGF), and nitric oxide. The increase in catabolic mediators due to excessive weight load and adipose tissue, which is not accompanied by cartilage matrix production promotes chondrocyte apoptosis leading to cartilage erosion.[1,7–9,12–15]

Thinning cartilage can cause the bone to crack, forming small crevices on the bone surface, allowing synovial fluid to enter. However, these may form cysts and develop into osteophytes. This process alters the biomechanical strength of the joint, resulting in abnormal loading and articular damage.[16,17] The accumulation of degradation products in the cartilage leads to infiltration of inflammatory cells into the synovial fluid (synovium effusion), causing synovitis.[1,7,13] In compensation, the cartilage repaired by increasing synthesis of matrix macromolecules by chondrocytes, resulting in a phase of cartilage hypertrophy.[5]

The gold standard for obesity screening is dual-energy x-ray absorptiometry (DEXA), which assesses body fat percentage and adiposity, ≥25% in males and ≥35% in females.[18,19] A simpler way to diagnose obesity is through anthropometry. The body mass index (BMI) is currently the most common measure of obesity. However, BMI has limitations for determining the location and percentage of body fat as it cannot distinguish between accumulated fat mass, muscle mass, or other factors.[18] This means that individuals with a higher volume of fat mass but the same BMI may be considered equally healthy.

To address this issue, it requires an additional indicator that takes into account the volume of fat mass in its calculation to improve validity and accuracy using body shape index (BSI), which combines BMI and waist circumference[20], introduced by Krakauer. BSI can reflect visceral adiposity independently of BMI[21], and is more accurate in describing lipoprotein variability than BMI[22]. The combination of BSI and BMI increases a significant association with the incidence of metabolic syndrome[23], which is one of KOA risk factors.[1,7]

This research is still rare regarding the relationship between BSI and KOA and also unclear whether BSI or BMI has a greater influence on KOA. Therefore, this study aims to identify which indicators are more closely related to KOA, in order to facilitate their implementation in public health with the aim of reducing KOA risk factors.

METHOD

This research is an observational analytic with a case control design to assess the correlation of BSI and BMI with KOA. It has been approved by The Ethics Commission of Health Research, Faculty of Medicine, University of Jember. The study population comprised patients from the medical records of the Faculty of Medicine University of Jember's social service conducted on 18 June 2023 at Jenggawah Village, Jember Regency, who met the inclusion criteria. These criteria included patients with and without KOA who were willing to participate as respondents, with KOA symptoms in the last 6 months, and anthropometric data is available: weight, height, and abdominal circumference. Patients with congenital abnormalities and leg shapes, a history of knee trauma, prosthetic knees, vertebral injuries and surgeries, and pregnant patients were excluded.

The KOA clinical criteria used are based on the 1990 American College of Rheumatology which is also used by the Indonesian Rheumatological Association. These criteria consist of knee pain accompanied by three out of six factors: age >50 years, joint stiffness in the morning, crepitation, bony tenderness, bone enlargement, and non-warm sensation.[4] A sample of 30 KOA patients and 30 non-KOA patients were selected simple randomly [Figure 1].

The research instrument used was Omni Calculator, which calculated BSI based on anthropometric data: gender, age, weight, height, and waist circumference, obtained from medical records. The Omni Calculator for BSI was developed by Joanna Michałowska from Poznan University, based on Krakauer's (2012) research, which can be accessed at the link https://www.omnicalculator.com/health/a-body-shape-index. The body mass index and body shape index classification can be seen on Table 1.

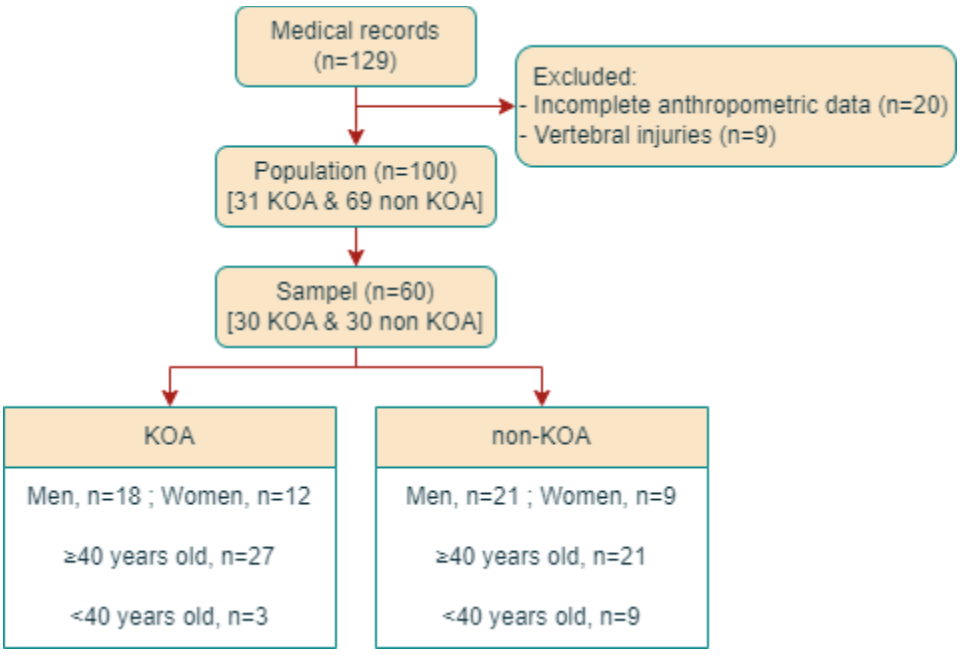

Figure 1. Flow chart of study participants

Each variable underwent univariate analysis to determine demographic characteristics of the sample. Bivariate analysis employed the Spearman Rank correlation test with a significance level of $p<0.05$ to determine the strength, direction, and existence of a relationship between the independent variables BSI and BMI and the dependent variable KOA.[24]

Table 1. Body mass index and Body shape index classification

Category	Range
Body Mass Index	
Very underweight	< 17,0
Underweight	17 - < 18,5
Normal	18,5-25,0
Overweight	> 25,0-27,0
Obese	> 27,0
Body shape index	
Low	< -0.868
Below average	-0.868 to -0.272
Average	-0.272 to +0.229
Above average	+0.229 to +0.798
High	> +0.798

Source: Ministry of Health of the Republic of Indonesia listed in the Minister of Health Regulation number 41 of 2014, Krakauer & Krakauer (2012) study.

RESULT

The majority of the KOA samples were male and aged ≥40, while non-KOA samples were also male and aged ≥40 (Figure 2).

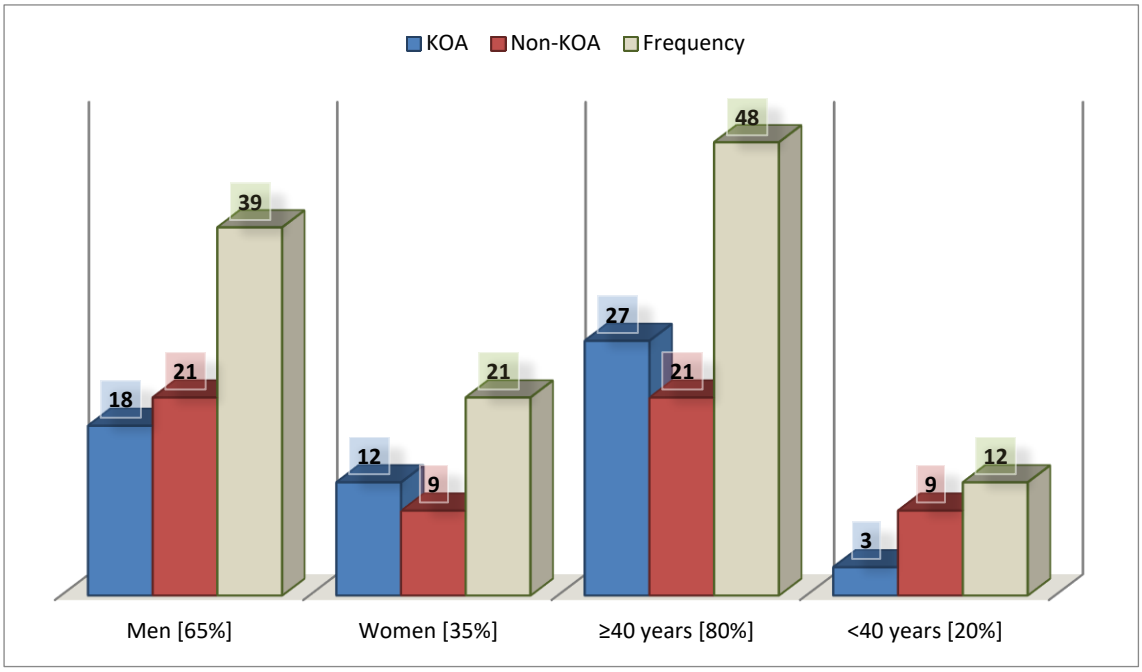

Figure 2. Samples Characteristics of KOA and Non-KOA

Results of Spearman Rank correlation test are presented in Table 2. The analysis showed a significant correlation between BSI and KOA (p=0.002) with a positive moderate strength (r=0.391), showing that KOA risk increases with BSI. The BMI and KOA showed a non-significant correlation (p=0.432); negative correlation (r= -0.103) which means the risk of KOA decreases as BMI increases; and very weak strength but this is not statistically significant.

DISCUSSION

This study looks at the association between BMI and BSI with the knee osteoarthritis (KOA) status. We found there is a moderate positive correlation between BSI and KOA, while non-significant weak negative association between BMI and KOA. Differences in the characteristics of previous studies may be attributed to variations in the intensity and type of physical activity between genders.[25] Women typically engage in low or moderate intensity, while men tend to engage in higher intensity. High intensity has been shown to harm knee joint and is associated with future OA progression.[26,27] It is worth noting that the higher incidence of KOA in men in this study may be attributed to the greater number of male participants.[28]

Table 2. Results of correlation analysis of BSI versus BMI with KOA

Variables	KOA	Non-KOA	Total	r	p
	f (%)	f (%)	f (%)		
Body shape index					
Low	2 (3.33)	11 (18.33)	13 (21.77)		0.002
Below average	2 (3.33)	3 (5.00)	5 (8.33)		
Average	6 (10.00)	6 (10.00)	12 (20.00)	0.391	
Above average	8 (13.33)	5 (8.33)	13 (21.77)		
High	12 (20.00)	5 (8.33)	17 (28.33)		
Body mass index					
Very underweight	0 (0.00)	0 (0.00)	0 (0.00)		0.432
Underweight	3 (5.00)	0 (0.00)	3 (5.00)		
Normal	15 (25.00)	15 (25.00)	30 (50.00)	-0.103	
Overweight	4 (6.77)	8 (13.33)	12 (20.00)		
Obese	8 (13.33)	7 (11.77)	15 (25.00)		

The sample was predominantly aged ≥40 years, consistent with meta- and cross-sectional analyses showing a linear increase in prevalence of symptomatic KOA with age >40.[29,30] Being aged >40 increases the risk of KOA 1.02-fold over those <40 years.[31] Aging contributes to OA through age-related inflammation, oxidative stress, and energy metabolism dysfunction.[32] Age-related pro-inflammatory mediators that may contribute to OA originate from adipose tissue in the infrapatellar fat pad around the knee joint, which increases with age. Additionally, they are produced locally within joint tissues, including chondrocytes and meniscus cells. Aging adipose tissue increases production of cytokines, such as interleukin-6 and tumor necrosis factor-a, chemokines and matrix-degrading enzymes, which damage the joint tissue.[33]

BSI is considered more effective than other anthropometry-based measures of adiposity because it can assess abdominal fat distribution. In contrast, BMI cannot recognize between fat mass and lean mass, and doesn't assess fat location. Additionally, waist circumference is distorted towards individuals of different body heights.[34–36] A high BSI result suggests that the waist circumference is larger than expected based on an individual's height and weight, indicating a central accumulation of body volume.[37,38]

BSI measurement can account for the complex nature of obesity, which requires the inclusion of general and abdominal fat indices, and is positively associated with visceral adiposity. Abdominal obesity is linked to cardiovascular risk and metabolic disease and is correlated with the degree of visceral obesity. Abdominal and visceral adiposity may promote more inflammatory factors and trunk load centered on the knee.[21,34,37,39] BSI values may indicate low skeletal muscle mass with increased visceral abdominal adiposity.[40] Currently, BSI is the only abdominal obesity index that is not influenced by the obesity paradox.[41]

Obesity causes KOA due to pathological changes in the entire knee joint structure, including abnormal loading on the joint, joint misalignment, and muscle weakness. Joint misalignment and muscle weakness dynamically influence each other and contribute to abnormal loading in obese individuals.[42] Excessive loading and increased adipose tissue can cause inflammation of the joint cartilage and metabolic imbalance, ultimately

leading to joint cartilage rupture.[8,43]

Adipose tissue can produce pro-inflammatory cytokines as well as adipokines like leptin and adiponectin. These regulate articular chondrocytes by increasing catabolic and inflammatory mediators, such as matrix-breaking enzymes. However, an increase in catabolic mediators without a corresponding synthesis of type 2 collagen and proteoglycans for matrix production leads to cartilage erosion.[1,13,43]

The study shows a positive and unidirectional correlation between BSI and KOA risk. This finding is consistent previous research, which indicates that the risk of cardiovascular disease (CVD) increases with higher BSI quartiles. BSI showed the strongest association of all predictors with all types of CVD. Severe obesity (BMI ≥40 kg/m^2) increases the risk of CVD by almost double, while BSI in categories Q2, Q3, and Q4 have a two, three, and eight times higher risk of CVD.[44] Another study revealed that lower and middle quintile ABSI-z score were associated with a decreased risk of mortality.[38]

Based on cohort studies, waist index (WSI) does not differentiate between high-risk and low-risk individuals in the categories of underweight (BMI<18.5 kg/m^2) or obesity (BMI≥30 kg/m^2), whereas the highest quartile of BSI can differentiate 18–39% of individuals in each BMI category, with a 22–55% higher risk of death. This suggests that BSI may be used to complement BMI in determining risk differences in the overweight and obese groups, as well as in the normal and overweight groups.[34]

Although in the above study BSI acted as a complement to BMI, this study demonstrates that BSI has a significant relationship with the incidence of knee OA and can assess population risk independently of BMI. This is corroborated by research which demonstrates that BSI can independently assess cardio-metabolic risk based on central obesity. Similar findings have been reported in other studies that have demonstrated the ability of BSI to predict fat mass index (FMI) and fat mass/fat-free mass (FM/FFM) ratio in women.[45] Furthermore, BSI has been shown to be capable of identifying visceral and sarcopenic obesity in overweight/obese adults who suffer from diabetes mellitus type 2.[23]

This study also found no significant relationship between BMI and KOA, possibly due to the limitations of BMI to distinguish between accumulated fat, muscle, or other mass, and doesn't determine the location of fat.[21] The BMI can only assess overweight (BMI >25) and not excess body fat. However, obesity affects KOA by causing fat accumulation that produces inflammatory factors, decreases muscle mass due to infrequent activity, increases knee load, or changes the biomechanics of the knee joint.[46,47] Other contributing factors may include unique demographic characteristics of patients in each region, such as socioeconomic conditions, anatomical trends, and physical activity, which require further study.[48]

The correlation between BMI and KOA is negative (inverse) due to the number of samples with overweight BMI was higher in non-KOA samples than in KOA samples, reaching twice as much. Additionally, there are KOA patients who have an underweight BMI, while non-KOA patients do not have an underweight or very underweight BMI. Based on data, patients who have an underweight BMI and KOA and those who have an overweight BMI and without KOA are typically >40 years old, suggests that is caused by age factor.[29,31]

Align with the increase of age, the percentage of body fat increases, leading to an increase in abdominal fat distribution.[32] In a cross-sectional study of type 2 diabetes mellitus patients, an inverse correlation was found between BMI and OA. BMI values decrease significantly as BSI values increase because BSI is inversely related to muscle mass independently of BMI and is related to fat-free mass as determined by bioelectrical

impedance analysis.[40]

The findings of this study demonstrate discrepancies with those of previous research. The findings indicate that while BSI has a significant, BMI has an insignificant relationship with KOA. This suggests that BSI may be a more accurate indicator of KOA risk factors than BMI. The risk of developing KOA increases with the BSI category. Further research with a wider scope and higher level of scientific evidence is recommended to ensure that BSI can replace BMI as an indicator of KOA risk factors and can be widely used by the public as a means of preventing KOA.

This study has limitations including the unbalanced distribution of gender and age of the sample because patients who come to social services tend to be male and older. Some medical records were incomplete especially on the additional risk factors for KOA, which precludes researchers from controlling for biases such as occupational or physical activities. Future studies should use a cohort design to validate whether BMI and BSI can predict the occurrence of knee OA. Expanding the sample size and research area will enhance the generalizability of findings.

CONCLUSION

The study concludes BSI has a significant positive correlation with the incidence of KOA, while BMI does not. To prevent KOA, individuals should manage their anthropometric growth, particularly body weight and waist circumference, as they are contributed to the accumulation of body fat, which is a risk factor for KOA. BSI should be used as other indicator beside BMI as anthropometric measure that should be routinely monitor to prevent the incident of KOA.

COMPETING INTEREST

The authors declare that they have no competing interests.

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AUTHOR'S CONTRIBUTION

AN, UE, and NSR designed the study and collected the data; AN analyzed the data and wrote the first draft of manuscript; YH and DAR proofread the draft; AN, UE, and NSR revised the draft. All authors approved the final manuscript.

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