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RESEARCH ARTICLE OPEN ACCESS

Impact of Long-Term Insulin Therapy on Body Composition and Metabolic Age in Type 2 Diabetes Mellitus Patients: A Cross-Sectional Study Using Bioelectrical Impedance Analysis

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Abstract:

Introduction: Patients with Type 2 Diabetes Mellitus (T2DM) are prone to changes in body composition, which are often characterized by excess visceral fat and metabolic aging. Long-term insulin use is known to influence weight and fat distribution, but its effect on metabolic age remains unclear.
To examine the relationship between Body Mass Index (BMI), visceral fat, metabolic age, and the duration of insulin therapy among patients with T2DM using Bioelectrical Impedance Analysis (BIA).

Methods: This cross-sectional study involved 199 adults with T2DM receiving insulin therapy at the Endocrine Outpatient Polyclinic. Anthropometric data and BIA-derived parameters were analyzed. Spearman's correlation assessed relationships among BMI, visceral fat, and metabolic age, while the Mann–Whitney U test compared body composition between short-term (<5 years) and long-term (≥5 years) insulin users.

Results: The majority of T2DM patients were in the obese category (69.3%). BMI showed significant positive correlations with both visceral fat ($\rho = 0.43$, $p < 0.001$) and metabolic age ($\rho = 0.41$, $p < 0.001$). Visceral fat was also weakly but significantly correlated with metabolic age ($\rho = 0.21$, $p = 0.002$). Patients on insulin for less than five years had slightly higher BMI than those on longer therapy (26.7 *vs* 25.6 kg/m^2; $p = 0.025$), while visceral fat and metabolic age did not differ significantly.

Discussion: These findings emphasize that adiposity plays a more dominant role than insulin exposure duration in determining metabolic aging and changes in body composition.

Conclusion: Higher adiposity, especially visceral fat, is strongly linked with accelerated metabolic aging in insulin-treated T2DM, whereas insulin duration has minimal effect. Nurses should facilitate routine body-composition monitoring using BIA to detect early metabolic deterioration and support individualized interventions targeting weight control and metabolic health.

Keywords: Body mass index, Visceral fat, Metabolic age, Diabetes mellitus, Insulin, Visceral adipose tissue.

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1. INTRODUCTION

Globally, diabetes mellitus is increasing significantly worldwide, with an estimated 589 million adults (20-79 years), 853 million in 2050 living with diabetes, 4/10 adults with diabetes undiagnosed, and 3.4 million deaths due to diabetes in 2024 [1], and 747,000 deaths occurred in 2021 [2]. Type 2 diabetes mellitus (T2DM) accounts for approximately 95% of cases, and represents about 5% type 1 diabetes mellitus (T1DM) [3].

Obesity is a major risk factor for insulin resistance and T2DM. Abdominal obesity, specifically visceral adipose tissue (VAT), is associated with a greater risk of developing T2DM [4], and obesity significantly increases the risk of age-related diabetes [5]. Changes in body composition are a defining feature of T2DM, marked by increased visceral fat and decreased lean mass that, together, exacerbate insulin resistance and metabolic health [6]. The accumulation of visceral fat, as opposed to subcutaneous fat, is significantly associated with hepatic insulin resistance, low-grade inflammation, and increased cardiometabolic risk. The compositional changes influence glycemic control and may accelerate biological aging through mechanisms such as oxidative stress, mitochondrial dysfunction, and chronic metabolic inflammation [7, 8]. Subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT) increased with age in females, and slightly decreased, while VAT remained stable in males [9].

Metabolic age, a metric derived from bioelectrical impedance analysis (BIA), has become a comprehensive indicator of metabolic and physiological aging, incorporating data on body composition, basal metabolic rate, and cellular integrity [10]. A metabolic age that exceeds chronological age has been recognized as an indicator of metabolic stress and insulin resistance, particularly in individuals with a presumably normal body mass index (BMI) [10, 11]. Recent studies have shown correlations between metabolic age and visceral adiposity, insulin resistance metrics, and glycemic control in type 2 diabetes mellitus [12, 13].

Long-term exposure to insulin could promote lipogenesis, reduce lipid oxidation [14], and lead to gradual weight gain, especially in individuals with insulin resistance. Long-term insulin administration is associated with elevated fat mass and altered fat distribution, notably the formation of visceral adipose [15]. Nonetheless, results remain contradictory, and few studies have investigated the impact of insulin therapy duration on body composition and metabolic age, as measured by BIA [16].

Since visceral fat and metabolic age are both key indicators of metabolic health, it is essential to understand their interactions under prolonged insulin exposure. This study investigated the effects of prolonged insulin therapy on body composition, particularly visceral fat, and metabolic age in individuals with T2DM, using bioelectrical impedance analysis (BIA) as an effective, non-invasive method for evaluating body composition.

2. METHODOLOGY

2.1. Study Design and Participant

This cross-sectional study was conducted at the Endocrine Outpatient Clinic, Zainoel Abidin Hospital in Banda Aceh, Indonesia. A medium effect size with a power of 0.8, a confidence level of 95%, and an alpha of 0.05 was used to determine the sample size. A total of 199 adults with T2DM receiving insulin therapy were enrolled using a consecutive sampling approach.

Inclusion criteria were: (1) diagnosis of T2DM based on the American Diabetes Association criteria [17], 2) current use of insulin therapy for ≥6 months, and (3) willingness to provide informed consent. The exclusion criteria were as follows: (1) pregnancy or lactation; (2) comorbid conditions that impact fluid or fat distribution, such as chronic kidney disease stage 5, liver cirrhosis, or heart failure; (3) use of medications that affect body composition, including glucocorticoids, SGLT2 inhibitors, or GLP-1 receptor agonists; and (4) incomplete anthropometric or BIA data.

2.2. Clinical and Anthropometric Assessment

Demographic and clinical data, such as age, sex, duration of diabetes, and duration and type of insulin therapy, were obtained from medical records. Height and body weight were measured using a stadiometer and calibrated digital scale, and body mass index (BMI) was calculated as weight (kg)/height^2 (m^2).

2.3. Body Composition Analysis

Bioelectrical impedance analysis (BIA) was performed under standardized conditions: in the morning, after an overnight fast, and after bladder emptying. The BIA device used the InnerScan Dual, Tanita brand (RD-953). Parameters measured by BIA include visceral fat level and metabolic age. Metabolic age refers to the metabolic state of the body, based on basal metabolic rate (BMR) and physiological health indicators, and provides an assessment of biological age. Metabolic age is computed using a formula that includes height, weight, age, and BMR. Visceral fat and metabolic age values were recorded as continuous variables for analysis. Higher metabolic age relative to chronological age was interpreted as accelerated metabolic aging [1, 2].

2.4. Statistical Analysis

Data were analyzed using SPSS. Spearman's rank correlation (ρ) was used to assess the relationships among BMI, visceral fat, and metabolic age. Mann–Whitney U tests were applied to compare body composition parameters between insulin duration groups (<5 years *vs* ≥5 years). Statistical significance was defined as $p < 0.05$ (two-tailed).

2.5. Ethical Consideration

This study was approved by the Ethical Review Committee (ERC) of the Faculty of Nursing, Universitas Syiah Kuala, Indonesia, under ERC letter 035/EA/FK-RSUDZA/2023.

3. RESULTS

3.1. Demographic Data

Sociodemographic data of the respondents, including age, gender, highest level of education, occupation, having diagnosed DM, and complications due to diabetes, are shown in Table 1.

Table 1. Sociodemographic and clinical characteristics of respondents (n = 199).

Variable	Category	Frequency	Percentage
Age (Mean ± SD)	-	(60 ± 9.5)	-
Gender	Male	85	42.7
	Female	114	57.3
Last education	Elementary school	21	10.5
	Secondary school	27	13.6
	High school	61	30.7
	College	90	45.2
Occupation	Unemployed	68	34.1
	Housewife	11	5.5
	Government employee	25	12.4
	Entrepreneur	29	14.4
	Farmer/labourer/fisherman	4	2.0
	Retired	61	30.6
Duration of diabetes (years)	(Mean ± SD) <5	(11.2 ± 7.1) 53	26.6
	5-10	63	31.7
	>10	83	41.7
Insulin types	Prandial only	78	39.2
	Basal only	31	15.6
	Mixed	90	45.2
Duration of insulin therapy	< 5 years	105	52.8
	≥ 5 years	94	47.2
Average insulin doses	Prandial only	10.5 IU	-
	Basal only	12.9 IU	-
Complication	None	9	4.6
	Hypertension (HT)	25	12.6
	Decreased vision (DV)	29	14.5
	Heart disease (HD)	5	2.5
	Stroke	2	1.0
	Other combinations*	129	64.8

Note: *Other combinations: Respondents with ≥2 complications (*e.g.* HT + DV, HT + HD).
IU = International unit.

Table 1 shows the sociodemographic and clinical characteristics of the participants. The mean age was 60.0 ± 9.5 years, and 57.3% were female. Most participants had completed high school or higher education (76.0%), with the predominant occupational category being unemployed (34.2%). The mean duration of diabetes was 11.2 ± 7.1 years; 41.7% of participants had diabetes for over ten years. Regarding complications, 64.8% of participants presented with multiple comorbidities, most commonly hypertension and diabetic retinopathy, followed by isolated hypertension (12.6%). The majority of respondents used mixed insulin (45.2%), with the average prandial and basal insulin doses being 10.5 IU and 12.9 IU, respectively, and 47.2% of respondents had been on insulin therapy for≥ 5 years.

Table 2 presents the cross-tabulation between BMI and visceral fat categories. Progressive increases in visceral fat were observed with increasing BMI. All underweight participants (100%) had normal visceral fat, while the obese category was dominated by respondents with high and very high visceral fat (35.5% each category). The Chi-square test demonstrated a significant association between BMI and visceral fat level ($\chi^2 = 11.12$, p-value = 0.011), indicating that higher BMI categories were consistently related to higher visceral fat.

Table 3 illustrates the distribution of metabolic age according to BMI classification. A clear pattern was observed, with the proportion of participants showing accelerated metabolic age increasing markedly from the normal BMI category (14.3%) to the overweight (28.5%) and obese (60.8%) groups. The Chi-square analysis confirmed a significant association between BMI and metabolic age category ($\chi^2 = 22.84$, p-value < 0.001). This finding indicates a tendency that the higher the BMI, the greater the proportion of respondents experiencing accelerated metabolic age.

To complement the categorical distribution illustrated in Tables 2 and 3, continuous analyses were performed to quantify the strength of association among BMI, visceral fat, and metabolic age, and to evaluate differences by insulin therapy duration.

Table 4 shows a significant positive correlation between BMI and both visceral fat ($\rho = 0.430$, p-value < 0.001) and metabolic age ($\rho = 0.409$, p-value < 0.001). This suggests that elevated BMI values are linked to greater visceral adiposity and an increased metabolic age. Statistical significance (p-value < 0.001) indicates there was a correlation between BMI and visceral fat. The clinical significance is that, even though BMI is related to visceral fat, other factors are more important in determining it. BMI is used as an initial screening tool to identify those at risk of having high visceral fat.

Visceral fat demonstrated a significant, albeit weaker, correlation with metabolic age ($\rho = 0.214$, p-value = 0.002). These findings suggest that body composition, particularly adiposity, contributes substantially to metabolic aging among insulin-treated patients with type 2 diabetes mellitus.

Table 5 presents a comparison of body composition and metabolic age by duration of insulin therapy. Patients receiving insulin for less than five years demonstrated a notably higher median BMI (26.7 *vs*. 25.6 kg/m^2, p-value = 0.025) in comparison to those treated for equal or over five years. No significant differences were found in visceral fat (p-value = 0.315) or metabolic age (p-value = 0.767) between the two groups. The findings suggest that prolonged insulin use may be associated with slight weight loss, whereas visceral fat accumulation and metabolic aging are likely influenced by other metabolic and lifestyle factors beyond insulin exposure alone.

Table 2. Distribution of respondents based on BMI and visceral fat (n = 199).

BMI	Visceral Fat			*p*-value
	Normal	High	Very High	
Underweight	2 (100%)	0 (0%)	0 (0%)	0.011
Normal	16 (57.1%)	10 (35.7%)	2 (7.1%)	
Overweight	14 (45.2%)	8 (25.8%)	9 (29.0%)	
Obese	40 (29.0%)	49 (35.5%)	49 (35.5%)	
Total	72 (36.2%)	67 (33.7%)	60 (30.1%)	

Table 3. Distribution of respondents based on BMI and metabolic age (n=199).

BMI	Metabolic Age			*p*-value
	Decelerated (%)	Accelerated (%)	Total	
Underweight	2 (100)	0 (0)	2	<0.001
Normal	24 (85.7)	4 (14.3)	28	
Overweight	23 (74.2)	8 (25.8)	31	
Obese	54 (39.1)	84 (60.9)	138	
Total	103 (51.8)	96 (48.2)	199	

Table 4. Spearman correlation among BMI, visceral fat, and metabolic age (n=199).

Variable Pair	Spearman's ρ	*p*-value
BMI - Visceral fat	0.430	<0.001
BMI - Metabolic age	0.409	<0.001
Visceral fat - Metabolic age	0.214	0.002

Table 5. Comparison of body composition and metabolic age by duration of insulin therapy.

Parameter	< 5 years (n = 105) Median (IQR)	≥ 5 years (n = 94) Median (IQR)	*p*-value (Mann-Whitney U)
BMI (kg/m^2)	26.7 (23.9–29.4)	25.6 (23.4–28.6)	**0.025**
Visceral fat (level)	12.0 (8.3–15.0)	10.5 (8.0–14.2)	0.315
Metabolic age (years)	59 (52–66)	60 (54–66)	0.767

These analyses indicated an ongoing association among adiposity, visceral fat accumulation, and accelerated metabolic aging, whereas the impact of insulin therapy duration was comparatively limited. The findings emphasize an association between long-term metabolic alterations and body composition in individuals with type 2 diabetes mellitus.

4. DISCUSSION

The global prevalence of both obesity and T2DM has been rising worldwide. Obesity, highlighting a key factor driving this association, is characterized by body composition, particularly the accumulation of visceral fat, which is strongly associated with insulin resistance and the pathogenesis of T2DM [18]. Insulin resistance is an important pathogenic component of metabolic diseases, such as T2DM [6], and increased BMI, as reflected in a high waist circumference, is associated with a higher prevalence of insulin resistance [6].

The mean age of participants in this study was 60 ± 9.5 years, with females predominating (57.3%). This finding aligns with national and international epidemiological data indicating a higher prevalence of type 2 diabetes mellitus (T2DM) among middle-aged and older adults, particularly women [19, 20]. The greater proportion of women in this cohort may reflect longer life expectancy and higher health-care utilization rates among females with chronic diseases. A substantial proportion of participants (34.2%) were unemployed or retired, which may be associated with reduced physical activity and metabolic decline contributing to insulin resistance. The mean duration of diabetes in this study was 11.2 ± 7.1 years, indicating a population with long-standing disease and likely cumulative exposure to metabolic complications. Nearly two-thirds of participants (64.8%) presented with multiple comorbidities. These demographic characteristics contributed to the metabolic results seen in subsequent study findings. The higher visceral fat and accelerated

metabolic age observed in this study may be due to a combination of factors, including advanced age, female predominance, limited physical activity, and other comorbidities. Therefore, the metabolic aging patterns observed in this study likely reflect the combined influence of lifestyle and sociodemographic factors, as well as the direct effects of insulin therapy.

The respondents in this study used prandial-only, basal-only, or mixed insulin regimens. The majority of respondents used mixed insulin (45.2%) with an average insulin dose of 12.9 IU. There was a significant relationship between BMI and visceral fat, as well as between BMI and metabolic age. The proportion of participants with high or very high visceral fat increased progressively with higher BMI categories, consistent with the established role of visceral adiposity as a major determinant of insulin resistance [13, 16]. It also implied that higher adiposity is associated with greater visceral fat accumulation and accelerated metabolic aging. The findings support existing evidence that excess adiposity, especially visceral fat, is central to insulin resistance, chronic inflammation, and age-related metabolic dysfunction in T2DM patients [7, 12].

The possible mechanism by which higher BMI and both visceral fat and metabolic age are associated with each other can be explained by the metabolic consequences of adipocyte hypertrophy and ectopic fat deposition. Visceral adipose tissue acts as an active endocrine organ, which releases free fatty acids and pro-inflammatory cytokines such as IL-6 and TNF-α, then impairs insulin signaling and promotes systemic oxidative stress [21, 22]. This association has been reported in a large population-based study, indicating that visceral adipose tissue, rather than subcutaneous fat, is the strongest predictor of diabetes prevalence and insulin resistance across sex and racial/ethnic groups [23], and that visceral adiposity index and visceral body fat show a positive correlation.

These processes accelerate cellular senescence, mitochondrial dysfunction, and the accumulation of advanced glycation end products (AGEs), all of which contribute to the phenomenon often referred to as “metabolic aging.” The significant correlation between visceral fat and metabolic age in this study supports this mechanistic link.

Insulin therapy remains essential for achieving glycemic control in advanced T2DM; however, long-term insulin therapy exposure may be associated with body composition. Insulin is an anabolic hormone that promotes lipogenesis and inhibits lipolysis; thus, weight gain and increased adiposity are common during early insulin therapy [15, 23]. This study evaluated the impact of long-term insulin use on body composition and metabolic age in T2DM patients. Interestingly, in our study, patients who had received insulin therapy for more than 5 years had slightly lower BMI than those treated for a shorter duration, whereas visceral fat and metabolic age showed no significant differences. This pattern may reflect improved glycemic stability, dietary adaptation, or reduced insulin doses over time, leading to relative weight stabilization after initial treatment [6, 24]. Comparable findings have been reported in longitudinal studies, where weight gain plateaus after several years of optimized insulin titration and lifestyle modification [15]. The findings suggest that visceral fat accumulation and metabolic aging are likely influenced by other metabolic and lifestyle factors beyond insulin exposure alone [4].

From a clinical perspective, our findings emphasize the need to assess not only BMI but also visceral fat and metabolic age in T2DM patients, especially those on insulin therapy. BIA offers a simple, non-invasive means to quantify these parameters in routine practice, enabling clinicians to identify individuals with disproportionate visceral adiposity despite apparently stable body weight. Clinicians can identify patients with excess visceral adiposity despite stable body weight using BIA, a simple, non-invasive method.

5. LIMITATION

We recognize a few limitations of this study. Since the study design was cross-sectional, it is not possible to establish a causal link between the duration of insulin use and changes in body composition or metabolic age. BIA, as a useful and non-invasive tool for assessing body composition, can be affected by hydration status, which was not controlled in this study. Also, we did not quantify insulin dose, diet composition, or physical activity level, which could confound associations between insulin use and adiposity. Future longitudinal studies combining serial BIA with detailed metabolic profiling are needed to confirm the observed trends and explore mechanistic pathways linking insulin therapy, visceral adiposity, and metabolic aging.

6. NURSING IMPLICATION

It is important for healthcare providers to monitor body composition in T2DM patients who use insulin for long-term use, so that the risk of visceral adiposity accumulation and metabolic aging changes can be prevented and controlled.

CONCLUSION

This study demonstrates that higher BMI and visceral fat are significantly associated with accelerated metabolic age in T2DM, whereas the duration of insulin therapy has a limited effect on these parameters. These findings emphasize that adiposity plays a more dominant role than insulin exposure duration in determining metabolic aging and changes in body composition. Continuous monitoring of visceral fat and metabolic age using BIA, therefore, serves as a practical approach for risk stratification and long-term management of patients receiving insulin therapy.

AUTHORS’ CONTRIBUTIONS

The authors confirm contribution to the paper as follows: C.H.: Wrote the protocol, performed the data analysis, wrote the initial manuscript, and conducted the critical review of the manuscript; I.N.: Conducted the data collection and analyzed the data; A.Y.: Contributed to the

data analysis and critical review of the manuscript; D.W.: Contributed to the critical review of the manuscript; S.F.: Contributed to the data analysis and critical review of the manuscript. All authors have approved the final version of the article.

LIST OF ABBREVIATIONS

T2DM	=	Type 2 Diabetes Mellitus
BMI	=	Body Mass Index
BIA	=	Bioelectrical Impedance Analysis
T1DM	=	Type 1 Diabetes Mellitus
VAT	=	Visceral Adipose Tissue
SAT	=	Subcutaneous Adipose tissue

ETHICAL STATEMENT

This study was approved by the Ethical Review Committee (ERC) of the Faculty of Nursing, Universitas Syiah Kuala, Indonesia, Under ERC letter 035/EA/FK-RSUDZA/2023.

CONSENT FOR PUBLICATION

Not applicable.

STANDARDS OF REPORTING

STROBE guidelines were followed.

AVAILABILITY OF DATA AND MATERIALS

All data generated or analyzed during this study are included in this published article.

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None.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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