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Original article

Age-Group Comparison of the Cardio-Ankle Vascular Index in Hospitalized Patients with Type 2 Diabetes Mellitus: A Cross-Sectional Study

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ABSTRACT

【Objectives】Arterial stiffness is a strong predictor of cardiovascular events in patients with type 2 diabetes mellitus (T2DM); however, age-related changes in arterial stiffness among hospitalized patients with T2DM have not been fully elucidated. This study aimed to compare the cardio-ankle vascular index (CAVI) by age group in hospitalized patients with T2DM and to clarify the effects of aging.

【Methods】Patients with T2DM admitted for educational purposes or glycemic control were grouped into 5 age groups: 40–49, 50–59, 60–69, 70–79, and 80–89 years. The CAVI values at admission were compared between the groups. Furthermore, multivariate analysis (forced entry) was performed with CAVI as the dependent variable, and age, sex, body mass index (BMI), presence of stroke, T2DM duration, diastolic blood pressure (DBP), hemoglobin A1c, triglycerides, and estimated glomerular filtration rate as explanatory variables.

【Results】A total of 134 patients were included in the analysis, and the CAVI increased with age ($p < 0.001$). Multiple regression analysis identified age ($B = 0.068$, $t = 6.775$, $p < 0.001$), BMI ($B = -0.069$, $t = -3.705$, $p < 0.001$), and DBP ($B = 0.022$, $t = 2.841$, $p < 0.01$) as factors associated with the CAVI.

【Conclusions】Age was an independent factor associated with the CAVI in hospitalized patients with T2DM, with the CAVI increasing with age. These values were higher than those reported in healthy adults in previous studies across all age groups, with a larger difference from age 50 to 59 years and older. Interventions addressing arterial stiffness across all age groups are warranted.

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Introduction

Cardiovascular disease (CVD) remains the leading cause of death worldwide, with ischemic heart disease (IHD) and stroke accounting for the greatest burden. Deaths from IHD will reach approximately 9 million worldwide by 2021, making it the leading cause of death. Although mortality rates have declined slightly over the past 30 years, significant regional disparities persist and its prevalence will increase further with aging populations [1]. Meanwhile, both the number of stroke cases and deaths has increased, with respective increases of 44% and 70% between 1990 and 2021. Furthermore, these figures are projected to nearly double by 2050[2]. The common pathophysiological basis of these diseases is atherosclerosis, the progression of which is closely associated with an increase in arterial stiffness. Arterial stiffness is a predictor of cardiovascular events[3,4] and mortality[5,6], making its suppression critical for reducing CVD risk.

Type 2 diabetes mellitus (T2DM) has been implicated in increased arterial stiffness[7] and increases the incidence of CVD and mortality risk[8,9]. Therefore, assessing arterial stiffness in patients with T2DM is highly important for CVD prevention. The cardio-ankle vascular index (CAVI), an indicator of systemic arterial stiffness, is useful for assessment even in patients with T2DM because it is independent of blood pressure at the time of measurement[10].

Previous studies on healthy individuals have demonstrated that arterial stiffness gradually increases with age[11,12]. Furthermore, arterial stiffness also increases with age in individuals with CVD risk factors, such as impaired glucose tolerance, dyslipidemia, and hypertension[12]. However, the characteristics of age-related changes in arterial stiffness in patients with T2DM requiring hospitalization remain unclear. Patients with T2DM requiring hospitalization are presumed to have poor glycemic control and numerous complications that constitute cardiovascular risk factors. Therefore, changes in arterial stiffness with age may differ from those reported in previous studies.

Furthermore, the aging of patients with T2DM has recently become a major public health concern in developed countries, including Japan[13,14]. Therefore, understanding the age-related changes in arterial stiffness across a wide range of ages is necessary. Evaluating changes in arterial stiffness across a broad age range in patients with T2DM requiring hospitalization may aid in selecting exercise therapy interventions aimed at reducing the risk of CVD. Exercise therapy is a crucial treatment strategy in patients with T2DM. Physical therapists are required to develop intervention strategies that not only improve glycemic control and motor function but also target improvements in arterial stiffness, which directly impacts life expectancy. This study aimed to compare the CAVI values among patients with T2DM admitted for educational purposes or for glycemic control by age group and to clarify the relationship with aging.

Methods

1. Patients

This retrospective cross-sectional study enrolled patients with T2DM admitted to Tokushima Kensei Hospital for educational or blood glucose control hospitalization between 2015 and 2025. Individuals with an ankle-brachial index (ABI) < 0.9 were excluded because peripheral arterial disease may lead to falsely low (pseudo-normal) CAVI values[15]. Individuals for whom not all data were collected were also excluded. The patients were divided into 5 age groups based on their age at admission: 40–49, 50–59, 60–69, 70–79,

and 80–89 years.

2. Data collection

Data on age at admission, sex, body mass index (BMI), presence of stroke or IHD, use of insulin therapy, and duration of T2DM were collected from medical records. CAVI, ABI, heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were evaluated noninvasively using VaSera VS-1500 or VS-2500 (Fukuda Denshi Co., Ltd.). CAVI measurements were performed with the patients in the supine position at rest in a quiet room. The mean coefficient of variation of CAVI measurements was 3.8%, demonstrating good reproducibility[16]. The CAVI measurements were performed after confirming that stable heart sounds were detected. A low pressure of 30–50 mmHg was applied using four cuffs wrapped around the upper arm and ankle, which enabled the detection of pulse waves at these locations. Blood pressure was measured using an oscillometric method. The pulse wave velocity (PWV) was obtained by dividing the duration of the pulse wave propagating from the aortic valve to the ankle by the length of the vessel. CAVI was automatically calculated within this system using the following equation: $CAVI = a\,[(2\rho / PP) \times \ln(SBP / DBP) \times PWV^2] + b$. Here, PP indicates pulse pressure (SBP − DBP), ρ is blood density, and a and b are constants[16,17]. The mean CAVI value obtained from 5 to 6 pulse wave signals was adopted[17]. The mean values were calculated from the obtained left and right CAVI and ABI measurements and were adopted for analysis. For other secondary outcomes, we collected data on the following biochemical parameters related to blood glucose control, lipid metabolism, and renal function at admission: hemoglobin A1c (HbA1c), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides, creatinine, and estimated glomerular filtration rate (eGFR). The presence of hypertension and dyslipidemia was investigated based on biochemical parameters and medication status. Hypertension was defined as SBP ≥ 140 mmHg, DBP ≥ 90 mmHg, or receiving ongoing treatment for hypertension. Dyslipidemia was defined as LDL-C ≥ 140 mg/dL, HDL-C ≤ 40 mg/dL, triglycerides ≥ 150 mg/dL, or receiving ongoing treatment for dyslipidemia[18]. The presence or absence of insulin therapy was determined based on the patient's status immediately before admission.

3. Statistical analysis

Nominal variables are presented as n (%). For continuous variables, parametric data are shown as means and standard deviations, whereas non-parametric data are presented as medians and 25th–75th percentile values (interquartile ranges). To analyze for differences between groups, Fisher's exact test was used for nominal variables, and a one-way analysis of variance (ANOVA) or the Kruskal–Wallis test was used for continuous variables. Post hoc multiple comparisons (Bonferroni method) were performed. Spearman's rank correlation coefficient was used to investigate relationships with age. Additionally, multiple linear regression analysis was performed using the forced-entry (enter) method, with CAVI as the dependent variable. Explanatory variables were selected based on between-group differences and clinical relevance and were entered simultaneously into the model (age, sex, BMI, presence of stroke, DBP, duration of T2DM, HbA1c, triglycerides, and eGFR). Statistical analysis was performed using the EZR version 1.55[19], with the significance level set at $p <$

0.05.

In addition, relative increase rates (%) were calculated descriptively using mean CAVI values compared with healthy adults reported by Choi et al. (2013)[11]: (mean CAVI in the present study − mean CAVI in healthy adults) / mean CAVI in healthy adults × 100; these rates were calculated for the 40–49 to 70–79-year age groups.

4. Ethical considerations

This study was approved by the Ethics Committee of Tokushima Kensei Hospital (2024-Eth-05). The study details were publicly posted within the hospital, and participants were provided the opportunity to opt out.

Results

This study included 293 patients with T2DM. 17 patients with an ABI < 0.9 and 142 patients with missing data were excluded from the analysis. The final number of participants included in the analysis was 134 (Figure 1).

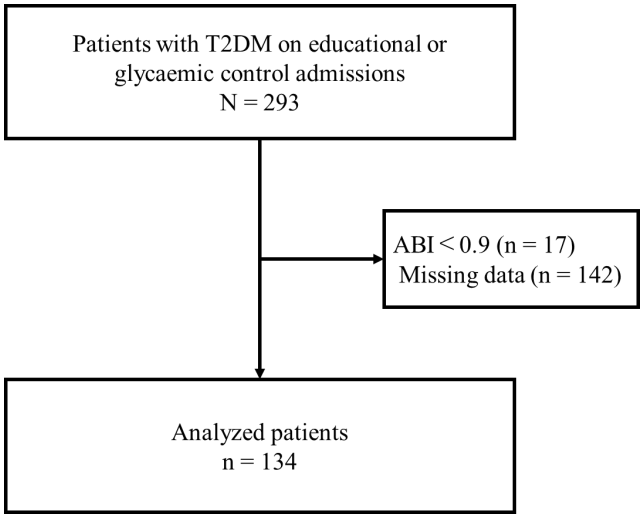

Figure 1. Flowchart showing patient screening and inclusion

T2DM, type 2 diabetes mellitus; ABI, ankle-brachial index

Regarding the basic characteristics (Table 1), BMI decreased with increasing age, and the 70–79-year age group had a significantly lower BMI than the 40–49-year age group ($p < 0.05$). The presence of stroke and hypertension as well as the duration of T2DM increased with age.

The ABI, HR, blood pressure, and biochemical parameters are presented in Table 2. DBP was significantly lower in the 70–79-year and 80–89-year age groups than in the 50–59-year age group ($p < 0.05$). Triglycerides levels in the 70–79-year and 80–89-year age groups were significantly lower than in the 50–59-year age group ($p < 0.05$). eGFR was significantly lower in the age groups 50–59 years and older than in the 40–49-year age group, and in the 80–89-year age group, it was significantly lower than that in the 50–59-year age group ($p < 0.05$).

The CAVI values (Figure 2) were 7.20 ± 0.86, 8.00 ± 0.87, 8.81 ± 1.15, 9.55 ± 0.98, and 10.11 ± 0.91 for the age groups 40–49, 50–59, 60–69, 70–79, and 80–89 years, respectively. ANOVA revealed significant differences between these values ($p < 0.001$).

CAVI and age showed a strong positive correlation ($r = 0.627$, $p < 0.001$; Figure 3). In the multiple regression analysis with CAVI as the dependent variable (Table 3), age ($B = 0.068$, $t = 6.775$, $p < 0.001$), BMI ($B = -0.069$, $t = -3.705$, $p < 0.001$), and DBP ($B = 0.022$, $t = 2.841$, $p < 0.005$) were independently associated with CAVI (adjusted $R^2 = 0.485$). The variance inflation factors for the included explanatory variables ranged from 1.1 to 1.8, indicating no significant multicollinearity issues.

The relative increase rates of CAVI values for each age group in this study compared with those reported by Choi et al. (2013) for healthy adults[11] were 3.75% for the 40−49-year age group, 8.70% for the 50−59-

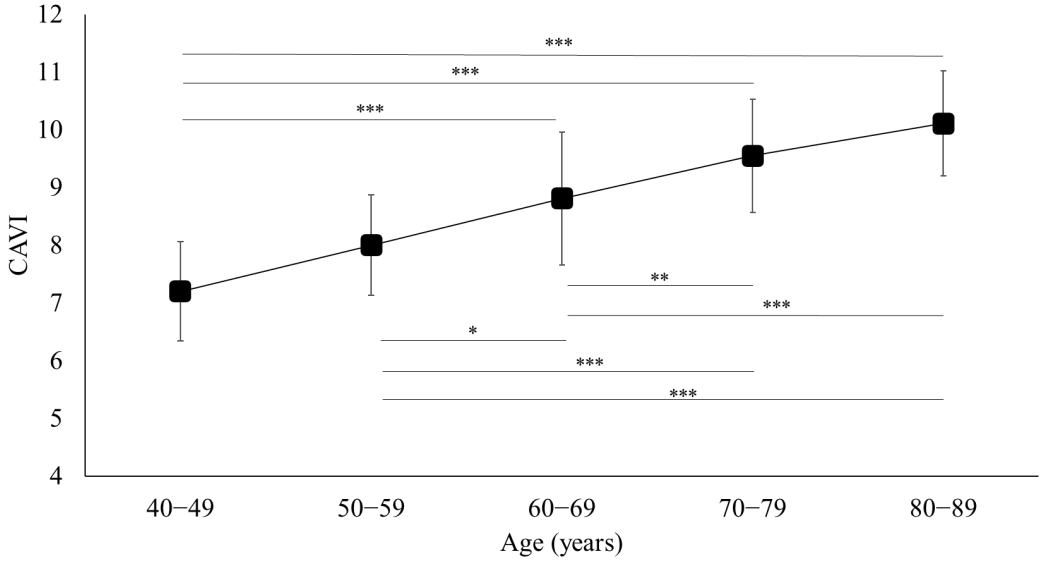

Figure 2. Intergroup comparison of CAVI

CAVI, cardio-ankle vascular index

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$ vs. 40–49 years; Bonferroni post hoc test.

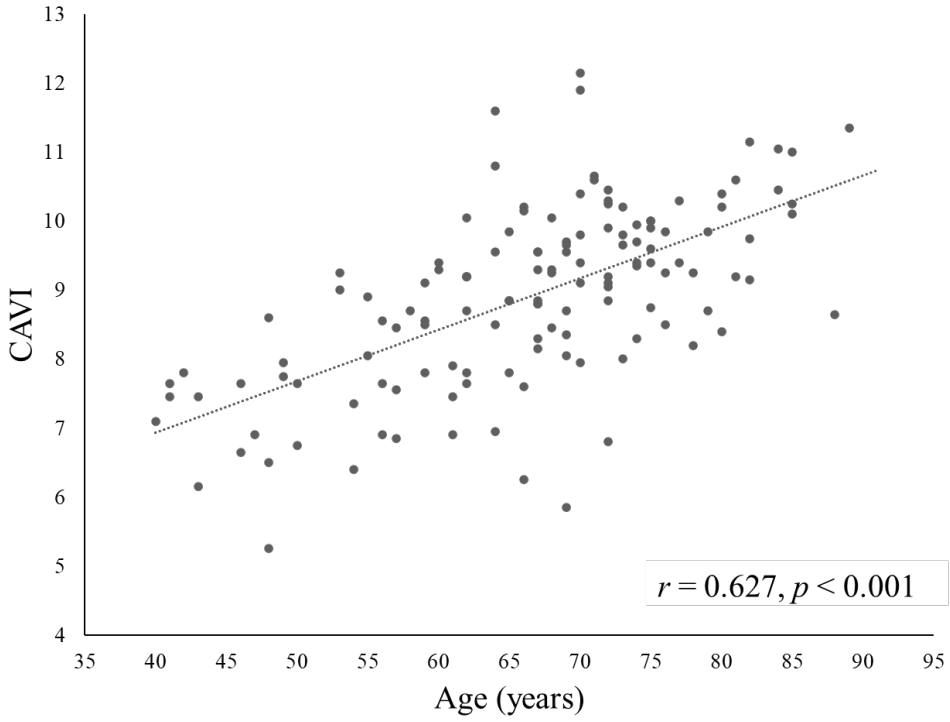

Figure 3. Correlation analysis of CAVI and age

CAVI, cardio-ankle vascular index. Applied Spearman's rank correlation coefficient.

Table 1. Intergroup comparison of basic characteristics

Parameters	Total	40–49 years	50–59 years	60–69 years	70–79 years	80–89 years	*p*-values
n (male/female)	134 (81/53)	14 (9/5)	19 (17/2)	44 (24/20)	42 (22/20)	15 (9/6)	0.055
BMI, kg/m^2	25.3 (22.2–29.0)	29.5 (25.8–31.3)	28.2 (23.0–30.0)	25.1 (22.7–29.0)	24.4 (21.3–26.7)*	25.1 (21.7–25.9)	0.008
Stroke, n (%)	15 (11.2)	0 (0)	1 (5.3)	4 (9.1)	4 (9.5)	6 (40.0)†	0.017
IHD, n (%)	22 (16.4)	0 (0)	4 (21.1)	7 (15.9)	6 (14.3)	5 (33.3)	0.171
Hypertension, n (%)	102 (76.1)	4 (28.6)	13 (68.4)*	36 (81.8)*	34 (81.0)*	15 (100.0)*	< 0.001
Dyslipidemia, n (%)	90 (67.1)	11 (78.6)	13 (68.4)	33 (75.0)	23 (54.8)	10 (66.7)	0.306
Insulin therapy, n (%)	32 (23.9)	4 (28.6)	5 (26.3)	13 (29.5)	7 (16.7)	3 (20.0)	0.664
Duration of T2DM, years	11.5 (3.0–19.0)	2.0 (0.0–6.8)	9.0 (1.0–16.5)	9.0 (3.5–15.5)	15.5 (10.0–24.0)*	15.0 (11.5–26.0)*	< 0.001

BMI, body mass index; IHD, ischemic heart disease; T2DM, type 2 diabetes mellitus.

Statistical analyses included Fisher's exact test, the Kruskal–Wallis test, and multiple comparisons (Bonferroni method).

*$p < 0.05$ vs. 40–49 years. †$p < 0.05$ vs. 40–49, 50–59, 60–69, and 70–79 years

Table 2. Intergroup comparisons of ankle-brachial index, heart rate, blood pressure, and biochemical parameters

Parameters	Total	40–49 years	50–59 years	60–69 years	70–79 years	80–89 years	*p*-values
ABI	1.11 ± 0.08	1.10 ± 0.08	1.11 ± 0.07	1.11 ± 0.07	1.11 ± 0.08	1.10 ± 0.09	0.982
HR, bpm	68.2 ± 12.2	72.1 ± 11.0	72.5 ± 14.0	67.6 ± 11.2	67.7 ± 10.7	62.1 ± 15.9	0.101
SBP, mmHg	134.0 (123.0–149.0)	133.0 (118.3–141.3)	134.0 (129.0–141.5)	130.0 (121.8–146.0)	140.5 (122.3–156.8)	135.0 (127.0–147.5)	0.633
DBP, mmHg	83.6 ± 11.1	84.6 ± 12.1	91.1 ± 12.4	84.0 ± 11.2	82.1 ± 9.1 §	76.2 ± 8.8 §	0.002
HbA1c, %	9.60 (8.60–11.20)	10.25 (8.13–11.55)	10.5 (9.10–11.30)	9.45 (8.70–11.20)	9.20 (7.98–10.38)	9.90 (8.65–11.90)	0.403
LDL-C, mg/dL	113.5 (94.0–136.8)	113.0 (104.8–127.0)	110.0 (95.5–149.5)	116.5 (93.3–138.3)	113.5 (96.5–135.0)	107.0 (87.0–127.0)	0.95
HDL-C, mg/dL	48.0 (40.0–56.0)	41.5 (36.8–47.8)	45.0 (33.5–53.0)	46.5 (40.8–55.3)	51.0 (42.3–59.0)	50.0 (44.0–65.5)	0.06
Triglycerides, mg/dL	134.0 (91.3–181.0)	156.5 (102.8–218.3)	192.0 (129.0–350.0)	139.5 (99.8–175.8)	118.5 (88.0–154.8) §	88.0 (76.5–115.0) §	< 0.001
eGFR, mL/min/1.73m^2	66.4 ± 21.5	88.7 ± 14.6	66.4 ± 23.9*	70.6 ± 21.5*	61.3 ± 17.3*	47.8 ± 13.4*†	< 0.001
Creatinine, mg/dL	0.80 (0.65–0.97)	0.69 (0.59–0.83)	0.89 (0.75–1.10)	0.73 (0.61–0.94)	0.83 (0.70–0.97)	0.93 (0.85–1.20)	< 0.001

ABI, ankle-brachial index; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; HbA1c, hemoglobin A1c; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate.

Statistical analyses included one-way analysis of variance, the Kruskal–Wallis test, and multiple comparisons (Bonferroni method).

*$p < 0.05$ vs. 40–49 years. §$p < 0.05$ vs. 50–59 years. †$p < 0.05$ vs. 60–69 years.

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Table 3. Multiple regression analysis with the cardio-ankle vascular index (CAVI) as the dependent variable

Explanatory variable	Regression coefficient (*B*)	95% confidence interval	*t*-value	*p*-value
Age	0.068	0.048 to 0.088	6.775	< 0.001
Sex (female)	−0.178	−0.529 to 0.173	−1.004	0.318
BMI	−0.069	−0.105 to 0.032	−3.705	< 0.001
Stroke	0.221	−0.314 to 0.757	0.817	0.415
DBP	0.022	0.007 to 0.038	2.841	0.005
HbA1c	−0.002	−0.087 to 0.083	−0.054	0.957
eGFR	0.001	−0.008 to 0.009	0.178	0.859
Triglycerides	0.001	−0.000 to 0.002	1.037	0.302
Duration of T2DM	0.012	−0.004 to 0.030	1.477	0.142

BMI, body mass index; DBP, diastolic blood pressure; HbA1c, hemoglobin A1c; eGFR, estimated glomerular filtration rate; T2DM, type 2 diabetes mellitus.

CAVI was used as the dependent variable, and age, sex, BMI, presence of stroke, DBP, HbA1c, eGFR, triglycerides, and duration of T2DM were entered as explanatory variables.

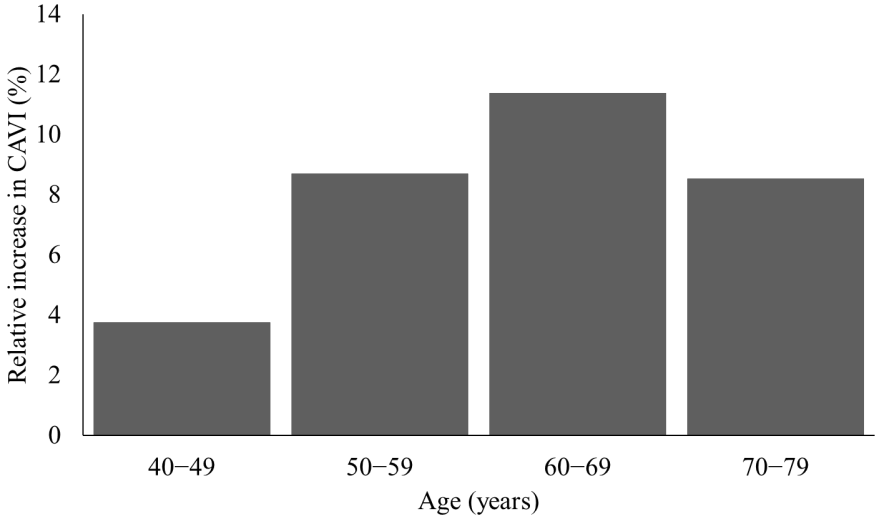

Figure 4. Relative increase in CAVI in hospitalized patients with T2DM compared to healthy adults

CAVI, cardio-ankle vascular index. Relative increase rate (%) = (CAVI of hospitalized patients with T2DM − CAVI of healthy adults) / CAVI of healthy adults × 100

year age group, 11.38% for the 60−69-year age group, and 8.52% for the 70−79-year age group (Figure 4).

Discussion

In this study examining the age-related changes in the CAVI among patients with T2DM admitted for educational purposes or glycemic control, CAVI increased with age. In a descriptive comparison with healthy individuals[11], the relative increase in CAVI was already evident in the 40–49-year age group and became more pronounced from the 50–59-year age group onward. To the best of our knowledge, this study is the first to clarify the characteristics of the CAVI according to age group in hospitalized patients with T2DM.

1. Association between CAVI and aging

This study identified a strong correlation between CAVI and age, and age was independently associated with an increased CAVI in the multiple regression analysis. Previous studies on healthy individuals and those at risk for CVD[11,12] have also reported an age-related increase in CAVI. The present study confirmed a similar trend in patients with T2DM. Furthermore, as all age groups exhibited higher levels than healthy individuals, the effects of chronic hyperglycemia associated with T2DM, accumulation of advanced glycation end products[20], oxidative stress[21], and endothelial dysfunction[22] associated with T2DM synergistically interact with aging and accelerate vascular wall stiffening. The regression coefficient (*B*) for age was 0.068 in this study and 0.048 in a previous study on healthy Asian individuals[11]. Thus, hospitalized patients with T2DM may exhibit an approximately 1.4-fold greater age-related increase in CAVI than healthy individuals. However, diabetes duration was not independently associated with CAVI, possibly because glycemic control may vary over the course of the disease and duration does not necessarily reflect cumulative glycemic burden. In addition, because hyperglycemia often precedes the diagnosis of T2DM, age may better capture the cumulative exposure relevant to *arterial* stiffening in the present study population.

2. Changes in other parameters

BMI, eGFR, and triglyceride decreased with advancing age. BMI decreased in the older age groups, with the 70–79-year age group showing significantly lower values than the 40–49-year age group. This may reflect reduced muscle mass and progression of sarcopenia. Multiple regression analysis revealed a negative association between BMI and CAVI, suggesting that atherosclerosis may progress in patients with T2DM with a low BMI. Previous studies have reported associations among sarcopenia, frailty, and increased arterial stiffness[23,24]. Thus, when implementing exercise therapy, a multifaceted approach encompassing blood glucose control, skeletal muscle mass, and arterial stiffness is necessary. Although eGFR and triglycerides decreased in the 70–79-year age group and older, no independent association between eGFR and triglycerides and arterial stiffness (CAVI) was confirmed. However, renal dysfunction contributes to the progression of arterial stiffness[25,26]. A Chinese cohort study has reported that triglycerides were significantly associated with carotid–femoral and carotid–radial PWV[27], and an independent association between CAVI and triglycerides has also been observed in the general European population[28]. Based on these previous studies, eGFR and triglycerides could certainly influence arterial stiffness; however, in the

study population of hospitalized patients with T2DM, these factors may more strongly reflect the effects of age and BMI.

DBP decreased in older age groups, peaking in the 50–59-year age group, consistent with the findings reported by Webb (2020)[29]. By contrast, in this study, DBP showed a positive association with CAVI after adjusting for age. Although CAVI theoretically does not depend on the blood pressure at the time of measurement, increased DBP reflects sustained vascular load and may promote structural remodeling, such as collagen deposition and elastin rupture[30]. Thus, among individuals of the same age, those with a relatively higher DBP may experience increased peripheral vascular resistance and structural stiffening owing to arterial wall remodeling. In hospitalized patients with T2DM, such as those in this study, these combined factors, in addition to chronic hyperglycemia, may help increase CAVI. The factors associated with CAVI included age, BMI, and DBP; however, the effect of age was particularly significant ($B = 0.065$, $t = 6.775$, $p < 0.001$). Therefore, age is the most influential factor and should be considered when determining the methodological aspects of physical therapy interventions during hospitalization.

3. Clinical significance

The study findings have important implications for physical therapists who prescribe exercise therapy for patients with T2DM. In a descriptive comparison with healthy individuals11), CAVI values were elevated in the 40–49-year age group, and the relative increase was greater in the 50–59-year age group and older. In the intergroup comparisons, ANOVA demonstrated a significant age-related increase. Bonferroni-adjusted post hoc tests showed significantly higher values in the 60–69-year age group and older than in the 40–49- and 50–59-year age groups, and significantly higher values in the 70–79- and 80–89-year age groups than in the 60–69-year age group. Therefore, arterial stiffness should be considered early when selecting interventions, and the necessity for this increases with age.

4. Limitations

This study had some limitations. As this was a retrospective cross-sectional study, causality could not be inferred. In addition, because the study population was limited to patients hospitalized for diabetes education or glycemic control, this was a single-center study, and sample sizes differed across age groups, the generalizability of these findings to the broader T2DM population should be interpreted with caution. Furthermore, the collected data were limited and did not include factors, such as skeletal muscle mass, exercise habits, physical activity level, and inflammatory markers. Therefore, these factors cannot be ruled out as confounding variables. Interventional studies are needed to optimize physical therapy to improve arterial stiffness in patients with T2DM.

5. Conclusion

This study examined age-related changes in CAVI in hospitalized patients with T2DM. CAVI increased with age, and age was an independent factor associated with elevated CAVI. In a descriptive comparison with healthy individuals11), the relative increase in CAVI was already evident in the 40–49-year age group and was greater from the 50–59-year age group onward. In intergroup comparisons within the present study population, CAVI values were significantly higher from the 60–69-year age group onward than in the 40–

49-year age group, with additional significant differences among the older age groups (≥60 years), indicating a stepwise increase with age. Therefore, interventions aimed at improving arterial stiffness are necessary at an early stage in patients with T2DM, and the importance of such interventions increases with age.

Conflict of Interest

The authors declare that they have no competing interests.

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