

Article

Respiratory Muscle Strength and Physical Function in Japanese Patients with Type 2 Diabetes

Hiroaki Kataoka ^{1,*}, Masayuki Tanaka ², Kenichi Deguchi ³, Kazuyuki Mori ³, Ryota Shinomiya ⁴, Fushi Higashine ⁵, Takanori Kiso ⁶, Shion Nagai ⁷ and Shinjiro Miyazaki ⁷

¹ Department of Human Sciences, Major of Physical Therapy, Kibi International University, 8 Iga-cho, Takahashi-City 716-8508, Japan

² Department of Physical Therapy, Faculty of Health Sciences, Okayama Healthcare Professional University, 3-2-18 Daiku, Kita-ku, Okayama-City 700-0913, Japan

³ Department of Rehabilitation Medicine, Tokushima Prefecture Naruto Hospital, 32 Kotani, Kurosaki, Muya-cho, Naruto-City 772-8503, Japan

⁴ Department of Rehabilitation, Tokushima Kensei Hospital, 4-9 Shimosuketo-cho, Tokushima-City 770-8547, Japan

⁵ Department of Rehabilitation, Kochi Medical School Hospital, 185-1 Kohasu, Oko-cho, Nankoku-City 783-8505, Japan

⁶ Hashimoto Kidney Clinic, 3-7-39-3 Matsunaga-cho, Fukuyama-City 729-0104, Japan

⁷ Rehabilitation Center, KKR Takamatsu Hospital, 4-18 Tenjinmae, Takamatsu-City 760-0018, Japan

* Correspondence: h.kataoka59@gmail.com; Tel.: +81-866-22-9495

Abstract

Background: Type 2 diabetes mellitus (T2DM) is associated with impaired skeletal muscle mass and function; however, respiratory muscle involvement has not been fully investigated, particularly in Japanese patients. This study aimed to evaluate respiratory muscle strength and identify its associated factors in Japanese patients with T2DM. **Methods:** This cross-sectional study included 55 patients with T2DM without respiratory disease. Maximal inspiratory pressure (P_Imax) and maximal expiratory pressure (P_Emax) were measured and expressed as percentages of the predicted values (P_Imax %pred and P_Emax %pred). Clinical variables, including age, sex, body mass index (BMI), diabetic polyneuropathy (DPN), skeletal muscle index (SMI), handgrip strength, and 6-min walk distance (6MWD), were assessed. Correlation and multiple regression analyses were performed to identify factors associated with P_Imax %pred. **Results:** P_Imax %pred was significantly lower than the predicted value (69.5% [95% CI: 63.1–75.9], $p < 0.001$), whereas P_Emax %pred did not differ significantly from the predicted value (95.6% [95% CI: 86.8–104.3]). P_Imax %pred was significantly associated with age, BMI, SMI, handgrip strength, and 6MWD. In multiple regression analysis, age, sex, BMI, and handgrip strength were independently associated with P_Imax %pred, whereas DPN was not. These findings remained unchanged after adjusting for smoking habits. **Conclusions:** Inspiratory muscle strength was selectively reduced in Japanese patients with T2DM and was independently associated with general physical characteristics and overall muscle strength. Inspiratory muscle weakness may reflect generalized skeletal muscle dysfunction rather than solely a neuropathic impairment. This assessment of respiratory muscle strength may provide additional insights into the physical function of patients with T2DM.

Keywords: type 2 diabetes mellitus; respiratory muscle strength; inspiratory muscle strength; physical function; handgrip strength



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1. Introduction

Type 2 diabetes mellitus (T2DM), a chronic disease that affects multiple organ systems, is associated not only with microvascular complications and neuropathy, but also with impairments in skeletal muscle mass and function [1–3]. In recent years, patients with T2DM have been recognized as a high-risk population for sarcopenia, which is closely associated with reduced muscle strength and physical performance [2,3]. However, most previous studies have focused on limb muscles, and the involvement of respiratory muscles has not been sufficiently investigated.

Respiratory muscles, including the diaphragm and intercostal muscles, are skeletal muscles that play essential roles in ventilation, exercise capacity, and physical function. Previous studies have reported reduced maximal inspiratory pressure and maximal expiratory pressure in patients with T2DM, suggesting respiratory muscle weakness [4,5]. In particular, diabetic polyneuropathy (DPN) has been proposed as a potential mechanism because it may affect the nerves innervating the respiratory muscles, and patients with DPN have been shown to exhibit more pronounced respiratory muscle impairment [6,7]. Furthermore, inspiratory muscle weakness is associated with autonomic dysfunction in this population [8].

Because the respiratory muscles are skeletal muscles, they may also be affected by systemic muscle impairments associated with T2DM. It is well established that patients with T2DM experience reductions in muscle mass and strength, which contribute to decreased physical function and exercise capacity [1–3]. However, the relationship between respiratory muscle strength, systemic muscle strength, and sarcopenia has not been sufficiently clarified. Therefore, it remains unclear whether respiratory muscle weakness in T2DM is primarily driven by neuropathic mechanisms or by generalized muscle weakness. Importantly, previous studies on respiratory muscle strength in patients with T2DM have mainly been conducted in Western populations, and evidence in Japanese patients remains limited. In Japan, studies on patients with diabetes have predominantly focused on limb muscle mass, muscle strength, and physical performance, whereas respiratory muscle function has rarely been systematically evaluated. Given that Japanese patients differ from Western populations in terms of body composition and the pathophysiology of T2DM, it is necessary to investigate respiratory muscle function specifically in this population.

Respiratory muscle strength is also clinically relevant because it may influence exercise tolerance and daily living activities. Moreover, inspiratory muscle training improves respiratory muscle strength and functional capacity in patients with T2DM [9,10], suggesting that respiratory muscle dysfunction may be modifiable. However, the characteristics of the patients who are more likely to exhibit respiratory muscle weakness have not been fully elucidated.

Therefore, the purpose of this study was to investigate respiratory muscle strength, including both inspiratory and expiratory components, in Japanese patients with diabetes and to identify the associated factors. Based on previous findings in Western populations, we hypothesized that respiratory muscle strength would be reduced in patients with diabetes, and that inspiratory muscles, primarily represented by the diaphragm, would be more impaired than expiratory muscles, owing to their different physiological characteristics [11]. We also examined the relative contributions of DPN and systemic muscle strength to respiratory muscle function.

2. Materials and Methods

2.1. Study Design

This was an exploratory cross-sectional study. Study subjects were recruited from among inpatients and outpatients of KKR Takamatsu Hospital (Takamatsu City, Kagawa Prefecture), Tokushima Prefecture Naruto Hospital (Naruto City, Tokushima Prefecture), Tokushima Kensei Hospital (Tokushima City, Tokushima Prefecture), Kochi Medical School Hospital (Nankoku City, Kochi Prefecture), and Hashimoto Kidney Clinic (Fukuyama City, Hiroshima Prefecture). Written informed consent was obtained from all participants. The research ethics committee of the Okayama Healthcare Professional University approved this study (approval number: 0104).

2.2. Study Subjects

Fifty-five T2DM patients without respiratory diseases were enrolled in this study. The inclusion criteria were as follows: (1) a diagnosis of T2DM, and (2) the provision of written informed consent. The exclusion criteria were as follows: (1) acute metabolic disorders; (2) acute or chronic musculoskeletal or cerebrovascular diseases; (3) severe limitations in activities of daily living; (4) dementia or suspected dementia; (5) a history of respiratory disease (e.g., chronic obstructive pulmonary disease, asthma, or interstitial lung disease); and (6) any condition deemed by the attending physician or principal investigator to make the patient unsuitable for participation.

2.3. Clinical and Laboratory Measurements

We collected data on sex, age (years), height (cm), body weight (kg), body mass index (BMI) (kg/m^2), duration of T2DM (years), medications, exercise, smoking habits, and laboratory test results. Fasting plasma glucose (mg/dL) and Hemoglobin A1c (HbA1c) levels (%) were measured using standard laboratory methods. Well-trained medical staff interviewed patients to assess their exercise and smoking habits. Additionally, information regarding the patients' exercise habits (defined as aerobic exercise [e.g., walking or jogging] for >30 min, three times per week over the preceding 6 months) [12]. DPN was diagnosed based on the guidelines of the Japan Diabetes Society [13]. As in our previous studies [14], DPN was defined as present if the patient had at least two of the following: (1) symptoms such as dysesthesia, pain, and/or loss of sensation; (2) decreased or absent Achilles tendon reflex; and (3) decreased vibratory sensation (≤ 10 s).

2.4. Measurement of Respiratory Muscle Strength, Skeletal Muscle Mass, Handgrip Strength, and Exercise Tolerance

Respiratory muscle strength was assessed by measuring maximal inspiratory (PI_{max}) and expiratory (PE_{max}) mouth pressures using a portable respiratory pressure meter (IOP-01, Kobata Gauge Mfg. Co., Ltd., Osaka, Japan) in accordance with the guidelines of the American Thoracic Society and European Respiratory Society [15]. Measurements were performed in a sitting position at residual volume for PI_{max} and at total lung capacity for PE_{max}. Two maximal efforts were made for each measurement, and the higher value was used for analysis. The participants were instructed to sustain the maximal pressure for at least 1 s. The measured values were expressed as percentages of the predicted values (PI_{max} %pred and PE_{max} %pred). The predicted PI_{max} and PE_{max} values were calculated using previously published reference equations for the Japanese population [16]. The prediction equations used to calculate PI_{max} %pred and PE_{max} %pred are provided in Supplementary Table S1. Because respiratory muscle strength is strongly influenced by age, sex, and body size, evaluation using population-specific predicted values has been recommended for the interpretation of respiratory muscle strength measurements [15].

Therefore, PImax and PEmax were primarily evaluated as percentages of predicted values in the present study.

Skeletal muscle mass was assessed using the skeletal muscle index (SMI). SMI was calculated as the appendicular skeletal muscle mass (ASM) divided by height squared ($SMI = ASM/height^2$ [kg/m²]). ASM was measured using bioelectrical impedance analysis with an InBody S10 (InBody Co., Ltd., Seoul, Republic of Korea), and measurements were performed according to a previous study [17]. Handgrip strength was measured using a handgrip dynamometer (T.K.K.5401; Takei Scientific Instruments Co., Ltd., Niigata, Japan), as previously described [18]. Handgrip strength was measured twice for each hand, and the highest value for each hand was used for analysis. Exercise tolerance was assessed using the 6-min walk test, and the 6-min walk distance (6MWD) was recorded according to standardized guidelines [19]. The test was conducted along a flat, straight corridor, and the participants were instructed to walk as far as possible within 6 min.

2.5. Statistical Analysis

Data were expressed as mean $\pm$ standard deviation. Normality was assessed using the Shapiro–Wilk test. To evaluate deviations from the predicted values, one-sample *t*-tests were performed to compare PImax %pred and PEmax %pred with the predicted values (100%). Associations between PImax %pred and the clinical variables were assessed using Pearson's or Spearman's correlation coefficients, as appropriate.

Multiple regression analysis was performed using PImax %pred as the dependent variable. Model 1 included age, sex, BMI, and DPN as independent variables (base model). Model 2 additionally included handgrip strength to examine the influence of overall muscle strength on inspiratory muscle strength. Smoking habits were considered a potential confounding factor affecting respiratory function; therefore, a sensitivity analysis was conducted by including smoking status (yes/no) in the regression model to assess the robustness of the results. In these analyses, $p < 0.05$ was considered statistically significant. All data were analyzed using the JMP 19.1.0 software (SAS Institute, Cary, NC, USA).

3. Results

The baseline characteristics of the study participants are summarized in Table 1. The proportion of males was 54.5%. The mean age was 66.7 ± 4.4 years, HbA1c was $8.3 \pm 1.9\%$, and the duration of diabetes was 14.0 ± 12.7 years. According to the assessment of respiratory muscle strength relative to predicted values, the PImax %pred was 69.5% (95% CI: 63.1–75.9), which was significantly lower than the predicted value of 100% ($p < 0.001$). In contrast, the PEmax %pred was 95.6% (95% CI: 86.8–104.3) and did not differ significantly from the predicted value.

The correlations between PImax %pred and clinical characteristics are shown in Table 2. PImax %pred was significantly associated with age, BMI, handgrip strength, SMI, and 6MWD but not with HbA1c.

The results of the multiple regression analysis for PImax %pred are presented in Table 3. In Model 1, age, sex, and BMI, but not DPN, were significantly associated with PImax %pred. In Model 2, age, sex, BMI, and handgrip strength, but not DPN, were independently associated with PImax %pred. To examine the robustness of these findings, a sensitivity analysis that included smoking habits was performed. The results remained unchanged, with age, sex, BMI, and handgrip strength consistently associated with PImax %pred.

Table 1. Clinical characteristics of type 2 diabetic patients.

Variable	Value
<i>n</i>	55
Age (years)	66.7 ± 4.4
Sex (male/%)	30 (54.5)
Height (cm)	160.0 ± 10.1
Body weight (kg)	64.8 ± 14.1
BMI (kg/m ²)	25.2 ± 4.4
Duration of diabetes (years)	14.0 ± 12.7
FPG (mg/dL)	164.1 ± 65.4
HbA1c (%)	8.3 ± 1.9
DPN (<i>n</i> /%)	29 (52.7)
Exercise habits (<i>n</i> /%)	16 (29.1)
Smoking habits (<i>n</i> /%)	11 (20.0)
SMI (kg/m ²)	7.03 ± 1.25
Handgrip strength (kg)	28.5 ± 8.5
6MWD (m)	363.0 ± 101.0
PI _{max} (cmH ₂ O)	59.0 ± 28.3
PI _{max} %pred (%)	69.5 ± 23.7
PE _{max} (cmH ₂ O)	68.8 ± 29.9
PE _{max} %pred (%)	95.6 ± 32.3

Values are presented as mean ± standard deviation or number (%). BMI, body mass index; FPG, fasting plasma glucose; DPN, diabetic polyneuropathy; SMI, skeletal muscle mass index; 6MWD, 6-min walk distance; PI_{max}, maximal inspiratory pressure; PE_{max}, maximal expiratory pressure; %pred, percent predicted.

Table 2. Correlations between PI_{max} %pred and clinical characteristics.

Variable	<i>r</i>	<i>p</i> -Value
Age	−0.78	<0.001
BMI	0.36	0.006
SMI	0.67	<0.001
Handgrip strength	0.79	<0.001
6MWD	0.49	0.001
Duration of T2DM	−0.34	0.012
HbA1c	0.12	0.392

BMI, body mass index; SMI, skeletal muscle mass index; 6MWD, 6-min walk distance; T2DM, type 2 diabetes mellitus.

Table 3. Multiple regression analysis for PI_{max} %pred.

Variable	Model 1 B (95% CI)	β	<i>p</i> -Value	Model 2 B (95% CI)	β	<i>p</i> -Value
Constant	102.1 (78.8 to 125.6)	-	<0.001	85.91 (62.0 to 109.8)	-	<0.001
Age	−0.832 (−1.04 to −0.63)	−0.494	<0.001	−0.757 (−0.95 to −0.56)	−0.450	<0.001
Sex (female)	−13.50 (−15.9 to −11.1)	−0.570	<0.001	−10.76 (−13.6 to −7.96)	−0.454	<0.001
BMI	0.840 (0.28 to 1.40)	0.157	0.004	0.642 (0.12 to 1.17)	0.120	0.018
DPN	1.324 (−1.22 to 3.87)	0.056	0.301	0.650 (−1.73 to 3.03)	0.028	0.586
Handgrip strength				0.580 (0.21 to 0.95)	0.209	0.003
R ²	0.89			0.91		

B, partial regression coefficient; β, standardized partial regression coefficient; BMI, body mass index; DPN, diabetic polyneuropathy.

4. Discussion

The present study demonstrated that inspiratory muscle strength, assessed as PI_{max} %pred, was significantly reduced in T2DM patients, whereas expiratory muscle strength (PE_{max} %pred) was relatively preserved. To our knowledge, this is one of the few studies to evaluate respiratory muscle strength in Japanese patients with T2DM using population-specific reference values. Furthermore, inspiratory muscle strength was independently

associated with handgrip strength rather than DPN, suggesting a closer relationship with generalized skeletal muscle dysfunction. These findings suggest preferential impairment of inspiratory muscle function in this Japanese cohort—a population in which data on respiratory muscle strength have been limited—and highlight the distinct behavior of inspiratory and expiratory muscle function in patients with T2DM. Previous studies have also reported reduced respiratory muscle strength in patients with T2DM [4–6].

One possible explanation for this selective reduction is the physiological and functional characteristics of the diaphragm, the primary inspiratory muscle. The diaphragm contains a high proportion of type I (oxidative) muscle fibers and is continuously active throughout life, making it highly dependent on oxidative metabolism [20]. Such characteristics may render it particularly vulnerable to the metabolic disturbances associated with T2DM, including mitochondrial dysfunction, oxidative stress, low-grade systemic inflammation, and other metabolic abnormalities [21]. These abnormalities may then impair muscle endurance and contractile function, particularly in muscles with high oxidative demand. Therefore, inspiratory muscles may be more susceptible to metabolic disturbances than the expiratory muscles, although direct evidence remains limited. This pattern is not unique to T2DM and has been reported in other chronic conditions. For example, structural and functional alterations in the diaphragm are well documented in chronic obstructive pulmonary disease, and inspiratory muscle dysfunction is a key feature of this disease [22]. Similarly, in patients with chronic heart failure and sarcopenia, reduced P_Imax has been identified as a clinically relevant indicator of respiratory muscle dysfunction [23,24]. These findings suggest that the diaphragm, due to its continuous activity and high metabolic demand, may represent a common site of vulnerability to chronic diseases. In contrast, expiratory muscles are recruited more intermittently and may therefore be less susceptible to these metabolic stresses, which could explain why P_Imax was reduced in the present study, whereas P_Emax remained relatively preserved. Taken together, these findings support the notion that inspiratory muscle weakness in T2DM may reflect diaphragm-specific vulnerability driven by metabolic and functional stress. However, the precise mechanisms underlying this selective impairment remain unclear, and further studies are required to clarify the relative contributions of metabolic abnormalities, neural factors, and muscle-specific characteristics.

Another important finding of this study was that P_Imax %pred was independently associated with age, sex, BMI, and handgrip strength. These results indicate that inspiratory muscle strength is influenced not only by disease-specific factors but also by general physical characteristics and overall muscle function. Age-related declines in muscle strength are well established, and sex differences in muscle mass and respiratory mechanics are also widely recognized [25]. BMI may reflect both body composition and the mechanical load on the respiratory system, which can influence respiratory muscle performance. The independent association between P_Imax %pred and handgrip strength suggests that inspiratory muscle weakness reflects generalized skeletal muscle dysfunction rather than localized impairment. Handgrip strength is a widely accepted marker of overall muscle strength and is commonly used to assess sarcopenia and physical frailty [26]. These findings are consistent with recent consensus statements on sarcopenia that emphasize muscle strength as a primary determinant of muscle function [27]. Given that respiratory muscles are also skeletal muscles, systemic muscle impairment in patients with T2DM may affect both the limbs and respiratory muscles in parallel. Previous studies have demonstrated that respiratory muscle strength is related to muscle mass and physical performance [28], supporting the notion that inspiratory muscle weakness may be part of a broader decline in skeletal muscle function. In addition, inspiratory muscle weakness in patients with T2DM may reflect not only diabetes-specific pathophysiological changes but also reduced physi-

cal activity and exercise tolerance, as well as generalized deconditioning. The observed associations among PImax %pred, handgrip strength, and 6MWD support the influence of overall physical function and neuromuscular performance. Although HbA1c was not significantly associated with PImax %pred, duration of T2DM showed a significant negative correlation with PImax %pred. This finding suggests that inspiratory muscle weakness may be related to the cumulative effects of long-term diabetes rather than current glycemic status alone. However, as this was a cross-sectional study with a relatively small sample size, the independent contributions of diabetes severity, disease duration, and physical activity-related factors could not be fully clarified.

Although DPN has been proposed as a potential mechanism underlying respiratory muscle dysfunction, its independent contribution was not demonstrated in this study. Previous studies have reported an association between DPN and respiratory muscle impairment [7,8]; however, these relationships may be influenced by confounding factors such as age, muscle strength, and overall physical condition. The effects of DPN may also depend on its severity or its specific clinical characteristics, which were not evaluated in detail in this study. Therefore, further investigations are warranted to clarify the contribution of DPN, particularly with respect to its severity and progression.

The results of this study have several important implications from a clinical perspective. Inspiratory muscle weakness appears to be a common feature in patients with T2DM, even in the absence of overt respiratory disease, suggesting that respiratory muscle function should be considered as part of a comprehensive assessment of physical function. The association between PImax %pred and handgrip strength indicates that simple, noninvasive measures of muscle strength may provide useful insights into respiratory muscle function, and that handgrip strength may serve as a practical surrogate marker when direct measurement is not feasible. Furthermore, interventions aimed at improving overall muscle strength, such as resistance or combined exercise programs, may have beneficial effects on respiratory muscle function. In addition, inspiratory muscle training has been shown to improve the respiratory muscle strength and functional capacity in patients with T2DM [9].

This study had several limitations. As this was a cross-sectional study, causal relationships could not be established. The sample size was relatively small, which may limit generalizability and increase the risk of overfitting. The severity of DPN was not assessed, which may have influenced the observed associations. A healthy control group was not included; therefore, respiratory muscle strength was evaluated relative to established Japanese reference values rather than through direct comparison with age-matched healthy individuals. In addition, pulmonary function testing was not performed; thus, the potential influence of subclinical ventilatory impairment on respiratory muscle strength could not be assessed. The study population consisted exclusively of Japanese patients; therefore, the findings may not be directly applicable to other populations. Finally, the timing and progression of inspiratory muscle weakness in T2DM patients remain unclear.

In conclusion, inspiratory muscle strength was selectively reduced in patients with T2DM and was independently associated with age, sex, BMI, and handgrip strength. The contribution of DPN remains unclear, and further research is needed to clarify its role. These findings suggest that inspiratory muscle weakness in this population may be associated with generalized skeletal muscle dysfunction.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/diabetology7070124/s1>, Table S1: Prediction equations for maximal inspiratory pressure (PImax) and maximal expiratory pressure (PEmax) in the Japanese population (Suzuki et al., 1997) [16].

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