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Unveiling the Link Between Low Handgrip Strength and Diabetic Foot Disease in Non-Geriatric Type 2 Diabetes Patients

ABSTRACT

Objective: Using non-geriatric type 2 diabetic patients with and without diabetic foot ulcers (DFUs), this study investigated the frequency of poor handgrip strength and examined the association between handgrip strength and diabetic foot disorder.

Methods: The study comprised a total of 525 patients with type 2 diabetes who visited the endocrinology outpatient clinic between August 2020 and June 2022; 242 of them had DFUs. Patients were selected according to inclusion and exclusion criteria. Data on medications used, as well as demographic and clinical information, were taken from hospital archives. The severity of ulcers was assessed using the Wagner score. Using a portable digital dynamometer, a test of handgrip strength was conducted. Low handgrip strength was defined as less than 16 kg for females and 27 kg for males.

Results: Patients with DFUs exhibited lower handgrip strength, older age, a longer duration of diabetes diagnosis, a lower body mass index, and higher fasting plasma glucose and hemoglobin A1c levels. Comorbidities such as peripheral artery disease and ischemic heart disease were more frequent in patients with DFUs and low handgrip strength. Diabetic foot ulcer frequency was 3.149 times higher among those with poor hand grip strength than those with good strength (odds ratio = 3.149; 95% CI, 2.118-4.672; $P < .001$).

Conclusion: Non-geriatric type 2 diabetes patients with DFUs had a higher frequency of low handgrip strength. Low handgrip strength was independently associated with the presence of DFUs. This study emphasizes the importance of addressing low handgrip strength as a potential risk factor in managing diabetic foot disease in non-geriatric type 2 diabetic patients.

Keywords: Diabetes, foot, handgrip, non-geriatric, ulcer

Introduction

The most frequent reason for non-traumatic lower limb amputations worldwide is diabetic foot disease. According to estimates, lower limb amputations are performed due to diabetes every 30 seconds in the earth, and most of those are said to take place after a foot ulcer.[1-3] Additionally, it is predicted that diabetic patients have a 19%-34% lifetime risk of developing a foot ulcer,[4,5] which is a significant source of mortality, morbidity, and financial burden on patients and healthcare systems.[1,6]

In daily tasks like gripping, twisting, or lifting objects, grip strength is essential. It has been utilized as a gauge for both nutritional status and everyday life activities.[7-9] One test to determine sarcopenia is the grip strength test, which measures muscular function.[10] As a result, grip power has been proposed as an aging biomarker.[11] It is a straightforward test that is quick, affordable, and non-invasive. Numerous studies on elderly patients have looked at their relationship with health outcomes like death, disability, and lengthened hospital stays.[12] Furthermore, nutritional conditions and frailty are reflected in grip strength.[7,13-15]

Interestingly, recent research found a link between handgrip strength and the healing of wounds.[16] When other characteristics were taken into account, patients with low handgrip strength had a 50% lower likelihood of having their wounds healed.[16] According to another study, elderly individuals with type 2 diabetes and diabetic foot conditions were more likely to have weak hand grips.[17] Additionally, in senior diabetic individuals, low grip strength was directly correlated with the Wagner score and strongly associated with the development of diabetic foot ulcers (DFUs).[17] However, sarcopenia and dynapenia may both be confounding in elderly patients as a result of aging.

Eren İmre[1]

Erdi İmre[2]

[1]*Department of Endocrinology and Metabolism, Dr. Ersin Arslan Education and Research Hospital, Gaziantep, Turkey*
[2]*Department of Orthopedics and Traumatology, Abdulkadir Yüksel State Hospital, Gaziantep, Turkey*

Corresponding author:
Erdi İmre
✉ erdiimre@gmail.com

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The link between grip force and diabetic foot disorder is not well understood and studied. This research seeks to determine the frequency of low grip force in patients who are not elderly and also have diabetic foot disorder and to evaluate the relationship between handgrip strength and the presence and severity of diabetic foot disorder in type 2 diabetic patients.

Materials and Methods

Study Design and Patients

In order to better understand the frequency of low grip force in type 2 diabetic patients with and without DFUs, as well as the connection between the 2, this single-center cross-sectional observational study was conducted. The study was carried out at the Gaziantep Dr. Ersin Arslan Training and Research Hospital Endocrinology and Metabolism Outpatient Clinics in Gaziantep, Turkey. The study comprised type 2 diabetes patients who visited the endocrinology outpatient clinic between August 2020 and June 2022. Based on the occurrence of DFUs, the patients were categorized into 2 groups. There were 242 patients with DFUs and 283 patients without DFUs, totaling 525 people. The following were the exclusion requirements: patients who are over 65 years old, have type 1 diabetes, are receiving hemodialysis, have a history of cancer, are using any dosage and duration of steroids during the year, have other neurologic diseases affecting muscle strength such as Parkinson's disease, have multiple sclerosis, have Alzheimer's disease or have mobility issues, have myopathy, or have quadriplegia as a result of a stroke.

Clinical Data

Clinical and demographic information was gleaned from patient records stored in our hospital's computerized data system. The presence of microvascular and macrovascular problems, the length of diabetes, the presence of concomitant conditions, and the drugs taken were all noted. The body mass index (BMI) was calculated using the factors of height and weight. As glycemic control parameters, this research used anthropometric data from the examination as well as data from our center's measurements of fasting plasma glucose (FPG) and hemoglobin A1c (HbA1c) values. The Modification of Diet and Renal Disease (mL/min/1.73m^2) equation [glomerular filtration rate (GFR) = 186 × (creatinine/88.4) − 1.154 × (age) − 0.203 × (0.742 if female) × (1.210 if black)] was used to compute the GFRs of the patients.

Ulcer severity was determined using the Wagner score (n = 5). When grading DFUs, the Wagner classification is most frequently employed. Meggitt created the Wagner method in 1976, and Wagner made adjustments in 1981.[15] Wagner scores divide DFUs into 6 classes (from G0 to G5) based on the depth of the ulcer.

Assessments of Muscular Strength

A handheld Smedley digital dynamometer (Baseline 12-0286 Digital Smedley Dynamometer, Fabrication Enterprises Inc., Elmsford, NY, USA) was used to measure handgrip strength in order to assess muscular strength. The mean value of all measures was used to calculate the score for each patient. Measures were collected in each hand 3 times, 1 minute apart. The National Health and Nutrition Examination Survey muscle strength/grip test procedure was used to apply the measurement.[18] Low handgrip strength was defined as less than 16 kg for females and 27 kg for males.[10]

Ethical Standards

The SANKO University's clinical research and ethical committee provided its approval to the study protocol (protocol 2020/317), which was carried out at the Gaziantep Dr. Ersin Arslan Training and Research Hospital Endocrinology and Metabolism Outpatient Clinics in accordance with the Declaration of Helsinki and the harmonization guidelines for good clinical practice. All participants verbally and in writing provided their informed permission.

Statistical Analysis

The Statistical Package for the Social Sciences Statistics software, version 22.0 (IBM Corp., Armonk, NY, USA) was accessed through our network and used for all statistical analyses. For continuous variables (FPG, weight, and lipid profile), data were evaluated as mean (+/–) SD, and for categorical variables (sex, etc.), they were evaluated as number (n) and percentage (%) values. The Kolmogorov–Smirnov test was used to examine the normal distribution. Based on the supposition of a normal distribution, an independent sample *t*-test or Mann–Whitney *U* variance analysis test was applied. The dependent sample *t*-test, or Wilcoxon test, was used to compare numerical data among dependent groups, depending on the assumption of a normal distribution. The category data were compared using the chi-square test.

The Spearman correlation analysis was used to determine the relationship between low handgrip strength and the numerical data (HbA1c, creatinine, FPG, etc.). To evaluate the relationship between 2 dichotomous categorical variables, Cramer's *V* is used. For dichotomous nominal data vs. ranks (ordinal), the rank–biserial correlation coefficient, r_{rb}, is utilized. The potential baseline risk factors for developing DFUs were examined using logistic regression. A *P* value of .05 or less was regarded as statistically significant.

Power analysis was calculated with the use of G-Power software, version 3.1.9.4, to determine the effective sample size required for our study. Based on data from a previous study,[17] an a priori power analysis for sample size estimation revealed that a sample size of 139 patients was required to reach 95% power at a 5% error rate and an effect size of 0.37. By taking into account the 5% error rate of the post hoc power analysis approach based on the existence of low handgrip strength in both groups encompassing 525 people, the power of the study was assessed as 99%.

Biochemical Measurements

Using hospital records, biochemical values from the previous month were documented. Total cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, and FPG were measured using colorimetric enzymatic techniques (Siemens Advia System, Deerfield, Ill, USA).

MAIN POINTS

- There is a significant association between low grip force and diabetic foot disease in non-geriatric type 2 diabetes patients, and diabetic foot ulcers show a substantial correlation with low handgrip strength.
- This study identifies potential influencing factors, such as age, body weight, and the presence of ischemic heart disease, which may contribute to the observed handgrip strength differences.
- The relationship between inflammation, vascular complications, and sarcopenia further highlights the importance of considering muscle health in managing diabetes patients with diabetic foot disorder.

Serum creatinine levels were determined using an ADVIA 1650 chemistry system from Siemens (USA), and HbA1c was determined using the immune-inhibition approach using an Advia 2400 chemistry system from Siemens Healthcare Diagnostics Inc.

Results

Clinical Characteristics and Laboratory Results of Patients

Table 1 lists patients' demographics, clinical traits, and biochemical findings concerning diabetes-related foot disease. Two-hundred forty-two of the 525 patients (46.1%) had a diabetic foot disease. Female patients made up 32.2% of DFU patients, and 62.2% of patients without DFUs. According to these statistically significant results, male individuals were found to have a higher risk of developing foot ulcers than females ($P < .001$). Older patients were observed to have diabetes-related foot ulcers ($P = .004$), and their diabetes had been present for much longer ($P < .001$) than those without foot ulcers. Lower BMI ($P < .001$), lower body weight ($P = .001$), higher FPG levels ($P = .001$), and higher HbA1c were all present in patients with foot ulcers ($P < .001$). Although individuals with foot ulcers had lower levels of HDL ($P < .001$) and low-density lipoprotein (LDL) cholesterol ($P = .001$), there was no discernible difference in triglyceride levels ($P = .988$) or non-HDL cholesterol ($P = .083$). Patients with foot ulcers had higher creatinine levels ($P < .001$), lower estimated glomerular filtration rate (eGFR) ($P = .011$), higher uric acid levels ($P = .004$), lower aspartate amino transferase (AST) levels ($P < .001$), lower alanine amino transferase (ALT) levels ($P < .001$), lower albumin levels ($P < .001$), higher white blood cell (WBC) counts ($P < .001$), and lower hemoglobin levels ($P < .001$) compared to those without foot ulcers.

Comorbidities

Individuals with diabetic foot disease were found to have a higher frequency of diabetic retinopathy ($P < .001$). Additionally, patients

Table 1. Demographics, Clinical Characteristics, and Biochemical Results of the Patients with Type 2 Diabetes According to the Existence of Diabetic Foot Disease

	Type 2 Diabetic Patients with Diabetic Foot Ulcers (n = 242)	Type 2 Diabetic Patients Without Diabetic Foot Ulcers (n = 283)	*P*
Gender (female), n (%)	78 (32.2)	176 (62.2)	**<.001****
Age (years)	56.00 (9.0)	55.00 (12.0)	**.004***
Duration of diabetes (years)	14.00 (9.0)	10.00 (10.0)	**<.001***
Diabetic retinopathy, n (%)	97 (40.08)	45 (15.9)	**<.001****
Complaints of neuropathy, n (%)	214 (88.4)	197 (69.6)	**<.001****
eGFR <60 mL/min/1.73 m^2	78 (32.23)	56 (19.78)	**.002****
Hypertension, n (%)	127 (52.5)	176 (62.2)	**.027****
Hyperlipidemia, n (%)	163 (67.4)	237 (83.7)	**<.001****
Coronary artery disease, n (%)	88 (36.4)	66 (23.3)	**.001****
Peripheral artery disease (%)	58 (24.0)	2 (0.7)	**<.001****
Smoking, n (%)	98 (40.5)	74 (26.1)	**.001****
Body mass index (kg/m^2)	29.41 (6.58)	32.43 (9.06)	**<.001***
Bodyweight (kg)	80.00 (19.0)	86.50 (21.5)	**.001***
FPG (mg/dL)	218.00 (164.0)	187.00 (131.30)	**.001***
HbA1c (%)	9.93 (3.50)	8.85 (4.00)	**<.001***
Triglycerides (mg/dL)	181.00 (124.0)	181.00 (132.0)	.988*
HDL cholesterol (mg/dL)	38.03 ± 14.70	45.03 ± 11.52	**<.001**
LDL cholesterol (mg/dL)	99.27 ± 34.65	110.64 ± 37.54	**.001**
Non-HDL cholesterol (mg/dL)	139.17 ± 44.53	146.69 ± 43.46	.083
Creatinine (mg/dL)	0.95 (0.44)	0.80 (0.28)	**<.001***
eGFR (mL/dk/1.73m^2)	73.29 ± 33.95	80.15 ± 25.60	**.011**
Uric acid (µg/L)	5.25 ± 1.64	4.82 ± 1.33	.004
AST (IU/L)	18.00 (9.00)	21.00 (11.00)	**<.001***
ALT (IU/L)	18.00 (10.00)	22.00 (16.00)	**<.001***
Albumin (g/L)	3.70 (0.90)	4.40 (0.50)	**<.001***
WBC (10^9/L)	10 125.00 (4855.00)	8195.00 (2830.00)	**<.001***
Hemoglobin (g/dL)	12.90 (3.10)	14.20 (2.50)	**<.001***
CRP (mg/L)	26.00 (75.95)	4.90 (8.00)	**<.001***
Low handgrip strength, n (%)	99 (40.9%)	51 (18%)	**<.001****
Handgrip strength (kg force)			
Female patients	16.57 (5.20)	19.69 (6.24)	**<.001***
Male patients	28.35 (11.04)	31.41 (9.51)	**<.001***

ALT, alanine amino transferase; AST, aspartate amino transferase; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; FPG, fasting plasma glucose; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; LDL, low-density lipoprotein; WBC, white blood cell.
* The Mann–Whitney *U*-test was performed (median/IQR).
**The Chi-square test was performed.

Table 2. The Comparison of the Drugs and Insulin Therapy According to Existence of Diabetic Foot Disease

	Type 2 Diabetic Patients with Diabetic Foot Ulcers (n = 242)	Type 2 Diabetic Patients Without Diabetic Foot Ulcers (n = 283)	*P*
Metformin, n (%)	164 (67.8)	232 (82.0)	**<.001**
DPP-4 inhibitor, n (%)	115 (47.5)	147 (51.9)	.336
SGLT-2 inhibitor, n (%)	44 (18.2)	92 (32.5)	**<.001**
GLP-1 analog, n (%)	0 (0.0)	12 (4.2)	**.001**
Sulfonylurea, n (%)	32 (13.2)	60 (21.)	.021
Thiazolidinedione, n (%)	20 (8.3)	59 (20.8)	**<.001**
Glinid, n (%)	5 (2.1)	14 (4.9)	.101
Statin, n (%)	38 (15.7)	68 (24.0)	**.022**
Fenofibrate, n (%)	22 (9.1)	47 (16.6)	**.013**
Acetyl salicylic acid, n (%)	121 (50.0)	94 (33.2)	**<.001**
ACE inhibitor/ARB use, n (%)	74 (30.6)	126 (44.5)	**.001**
Calcium channel blocker, n (%)	50 (20.6)	63 (22.3)	.482
Beta blocker, n (%)	57 (23.6)	69 (24.4)	.838
Insulin users, n (%)	188 (77.7)	141 (49.8)	**<.001**
Duration of insulin treatment (years)	8.00 (6.00)	8.00 (6.00)	.507
Total daily insulin dose (U/day)	50.00 (32.00)	48.00 (42.00)	.104

The Chi-square test was performed.
ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; DPP-4, dipeptidyl peptidase-4; GLP-1, glucagon-like peptide-1; SGLT-2, sodium glucose cotransporter-2.

with diabetic foot had greater frequencies of hyperlipidemia ($P < .001$), coronary artery disease ($P = .001$), peripheral artery disease ($P < .001$), complaints of neuropathy ($P < .001$), eGFR < 60 mL/min/1.73 m^2 ($P = .002$), and smoking ($P = .001$). However, those without diabetic foot disease had a higher frequency of hypertension ($P = .027$). Lower handgrip strength was seen in diabetic foot patients ($P < .001$). Additionally, compared to those without diabetic foot disease, both males and females with diabetic foot showed poorer handgrip strength ($P < .001$).

Medication Use

Drug and insulin therapy usage based on the presence of diabetic foot disease was evaluated and is listed in Table 2. Drug and insulin therapy usage based on the presence of diabetic foot disease was evaluated and is documented in Table 2. Use of metformin ($P < .001$), sodium glucose cotransporter-2 (SGLT-2) inhibitor ($P < .001$), glucagon-like peptide-1 (GLP-1) analog ($P = .001$), sulfonylurea ($P = .021$), thiazolidinedione ($P < .001$), statin ($P = .022$), fenofibrate ($P = .013$), angiotension-converting enzyme inhibitor, or angiotension receptor blocker ($P = .001$) were less common in the group of patients with diabetic foot disease. Between the 2 groups, there was no discernible difference in the use of beta-blockers ($P = .838$), calcium channel blockers ($P = .482$), dipeptidyl peptidase-4 (DPP-4) inhibitors ($P = .336$), or glinid ($P = .101$). Acetylsalicylic acid and insulin use were more frequent in the group of patients with diabetic foot disorder (both $P < .001$). However, the insulin treatment duration ($P = .507$) and the overall daily dose ($P = .104$) were comparable in both groups.

The composition of drugs was compared in non-geriatric diabetic patients with diabetic foot disease, considering grip force. Patients with lower and normal grip force had similar rates of metformin use ($P = .164$), sulfonylurea use ($P = .176$), thiazolidinedione use ($P = .813$), meglitinide use ($P = .999$), DPP-4 inhibitor use ($P = .436$), and SGLT-2 inhibitor use ($P = .738$). The fenofibrate and statin usage rates were also similar between these 2 groups ($P = .821$, $P = .596$), respectively.

Handgrip Strength Measurement

According to the grip force, patients with diabetic foot disorder had their demographic, clinical, and laboratory data evaluated and the results are documented in Table 3. Ninety-nine (40.9%) of the 242 individuals with DFUs had weak hand grips.

Older age ($P = .016$), lower body weight ($P = .012$), lower ALT levels ($P = .026$), higher creatinine levels ($P = .037$), lower eGFR values ($P = .009$), and a higher frequency of peripheral arterial disease ($P = .026$) and ischemic heart disease were all observed in patients with low handgrip strength.

Correlation and Regression Analysis

The presence of DFUs was found to be negatively connected with handgrip strength in both men and women ($P = .001$, $r = 0.355 r = 0.355$ and $P < .001$, $r = 0.262$, respectively). Lower grip force was found to be independently linked to diabetic foot disorder in all models of the multivariate analyses. The frequency of DFUs was 3.149 times higher among those with poor hand grip strength than those with good strength [odds ratio (OR) = 3.149; 95% CI, 2.118-4.672; $P < .001$) (Table 4).

Diabetic foot disease was also linked to low handgrip strength in all models of logistic regression analyses, and low handgrip strength was 3.144 times more frequent in patients with DFU (OR = 3.144; 95% CI, 2.118-4.672; $P < .001$) (Table 5).

Discussion

This study found that non-geriatric individuals with diabetic foot disease have a higher frequency of low grip force than those without the condition. Additionally, in the calculations of correlation and regression, low grip force was substantially linked to DFUs. The frequency of DFUs was 3.149 times higher among those with poor hand grip strength than those with good strength. The findings are consistent with earlier studies done on geriatric individuals.[19] Besides, diabetic foot disease was linked to low handgrip strength. Sarcopenia and

Table 3. Demographic, Clinical and Laboratory Parameters of Type 2 Diabetic Patients with Diabetic Foot Ulcer According to Handgrip Strength

	Patients with Low Handgrip Strength (n = 99)	Patients with Normal Handgrip Strength (n = 143)	*P*
Age (years)	57.00 (9.00)	55.50 (9.30)	**.016***
Gender (F/M), n	31/68	47/96	.889
Duration of diabetes (years)	12.00 (10.30)	15.00 (8.50)	.454*
Body weight (kg)	79.00 (15.00)	82.50 (20.00)	**.026***
Body mass index (kg/m^2)	29.29 (7.94)	29.70 (6.10)	.246*
Hypertension, n (%)	58 (58.6)	69 (48.3)	.119**
Hyperlipidemia, n (%)	68 (68.7)	95 (66.4)	.781**
Ischemic heart disease, n (%)	44 (44.4)	44 (30.8)	**.030****
Peripheral artery disease, n (%)	31 (31.3)	27 (18.9)	**.026****
Diabetic retinopathy, n (%)	41 (41.4)	56 (39.2)	.790**
Smoking, n (%)	35 (35.4)	63 (44.1)	.175**
AST (IU/L)	16.00 (9.00)	18.00 (9.00)	.118*
ALT (IU/L)	16.00 (10.00)	18.50 (9.00)	**.026***
FPG (mg/dL)	236.00 (179.00)	235.50 (160.80)	.852*
HbA1c (%)	10.25 (4.06)	10.04 (3.24)	.188*
Triglycerides (mg/dL)	180.50 (131.30)	175.00 (119.50)	.305*
HDL cholesterol (mg/dL)	39.01 ± 12.97	37.37 ±15.76	.437
LDL cholesterol (mg/dL)	101.86 ± 38.51	97.67 ± 32.13	.427
Non-HDL cholesterol (mg/dL)	140.40 ± 46.49	138.35 ± 43.36	.758
Creatinine (mg/dL)	0.98 (0.66)	0.92 (0.34)	**.037***
eGFR (mL/dk/1.73m^2)	66.46 ± 34.42	77.97 ± 32.93	**.009**
Uric acid (µg/L)	5.34 ± 1.68	5.18 ± 1.61	.499
WBC (10^9/L)	10 230.00 (5602.50)	9055.00 (3957.50)	**.001***
CRP (mg/L)	16.00 (45.98)	10.00 (35.00)	**.010***
Insulin users, n (%)	79 (79.8)	109 (76.2)	.511**
Duration of ınsulin treatment (years)	8.00 (6.00)	8.00 (6.00)	.488*
Total daily insulin dose (U/day)	50.00 (32.00)	50.00 (32.00)	.647*
Wagner score	Wg 1 = 3.0% Wg 2 = 20.2% Wg 3 = 45.5% Wg 4 = 26.3% Wg 5 = 5.1%	Wg 1 = 7.0% Wg 2 = 21.8% Wg 3 = 46.5% Wg 4= 22.5% Wg 5 = 2.1%	.056**

ALT, alanine amino transferase; AST, aspartate amino transferase; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; F, Female; FPG, fasting plasma glucose; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; LDL, low-density lipoprotein; M, Male; WBC, white blood cell; Wg, Wagner.
* The Mann–Whitney *U*-test was performed (median/IQR).
**The Chi-square test was performed.

Table 4. Variables Associated with Diabetic Foot Ulcer—Logistic Regression Results

Variable	Independent Variable	Odds Ratio/95% Confidence Interval	*P*
Univariate	Low handgrip strength	3.149 (2.118-4.672)	**<.001**
Age and gender adjusted	Low handgrip strength	2.770 (1.821-4.201)	**<.001**
	Age	1.034 (1.007-1.062)	**.015**
	Gender	3.391 (2.332-4.932)	**<.001**
Model 1	Low handgrip strength	2.066 (1.364-3.389)	**.001**
	Age	1.040 (1.010-1.071)	**.009**
	Gender	3.009 (2.011-4.504)	**<.001**
	Peripheral artery disease	30.303 (7.299-125.000)	**<.001**
	HbA1c	1.173 (1.075-1.280)	**<.001**
Model 2	Low handgrip strength	1.926 (1.069-3.472)	**.029**
	Age	1.033 (0.990-1.076)	.131
	Gender	2.650 (1.550-4.532)	**<.001**
	Peripheral artery disease	23.255 (3.039-166.666)	**.002**
	Hyperlipidemia	0.481 (0.251-0.919)	**.027**
	eGFR (mL/dk/1.73m^2)	1.006 (0.997-1.016)	.177
	CRP (mg/L)	1.040 (1.023-1.057)	**<.001**

CRP, C reactive protein; eGFR, estimated glomerular filtration rate; HbA1c, hemoglobin A1c.

Table 5. Variables Associated with Low Handgrip Strength—Logistic Regression Results

Variable	Independent Variable	Odds Ratio / 95% Confidence Interval	*P*
Univariate	Diabetic foot ulcer	3.144 (2.118-4.672)	**<.001**
Age and gender adjusted	Diabetic foot ulcer	2.777 (1.828-4.219)	**<.001**
	Age	1.054 (1.023-1.088)	.343
	Gender	1.223 (0.807-1.854)	**.001**
Model 1	Diabetic foot ulcer	2.190 (1.392-3.445)	**.001**
	Age	1.060 (1.026-1.095)	**<.001**
	Gender	1.164 (0.759-1.784)	.487
	Peripheral artery disease	2.358 (1.282-4.329)	**.006**
	HbA1c	1.078 (0.987-1.177)	.094
Model 2	Diabetic foot ulcer	1.958 (1.120-3.422)	**.018**
	Age	1.047 (1.009-1.086)	**.016**
	Gender	1.204 (0.745-1.946)	.449
	Peripheral artery disease	1.805 (0.975-3.333)	.060
	Hyperlipidemia	1.300 (0.771-2.197)	.324
	eGFR (mL/dk/1.73m^2)	1.009 (1.001-1.016)	**.021**
	CRP (mg/L)	1.000 (0.996-1.003)	.878

CRP, C reactive protein; eGFR, estimated glomerular filtration rate; HbA1c, hemoglobin A1c.

diabetic foot illness were related, according to another study published in the literature.[19] Several factors may play a role in pathophysiology, such as a decline in exercise and the ensuing decrease in muscle mass. Patients with diabetic foot disease have an enhanced inflammatory response, contributing to muscle mass loss. Previous cross-sectional investigations have demonstrated that systemic inflammation, fasting glucose, HbA1c, and hyperglycemia are inversely related to grip strength[20] and that in the general population, sarcopenia brought on by aging is associated with low-grade chronic inflammation.[21] The multimorbidity load was also correlated with grip strength.[22-24]

Moreover, hand grip strength is related to both WBC and C-reactive protein (CRP) levels.[25] This study revealed similar results to those in the literature. A study found that CRP as an indicator of acute inflammation is associated with maximal isometric handgrip strength in non-critically ill patients.[26] There is an association between cytokine production capacity and hand grip strength.[27]

In the elderly with type 2 diabetes, the frequency of low grip force was directly connected with the Wagner score in a prior study.[17] In addition to the apparent association, patients with DFUs who had weaker handgrips had considerably more significant rates of Wagner scores of 4 and 5 and lower rates of Wagner scores of 1 and 3.[17] However, this research's findings produced different results. The literature lacks evidence regarding the connection between handgrip strength and the severity of diabetic foot disorder.

This study also assessed patients with normal and lower muscular strength with diabetic foot disorders to identify potential influencing factors. Gender and the duration of diabetes between the 2 groups did not differ significantly, similar to a previous study performed on geriatric patients.[17] However, BMI did not vary significantly, and age was higher in non-geriatric individuals with low hand grip strength, contrary to a previous study with geriatric patients.[17] Patients with poor hand grips had significantly lower body weights, and this finding may be related to weight loss brought on by inflammation and starvation. Additionally, there were no significant differences in the frequencies of hypertension, hyperlipidemia, or symptoms related to diabetic neuropathy between these 2 groups. Non-geriatric patients with weak hand grip strength had increased frequency of peripheral artery disease, similar to geriatric patients,[17] and ischemic heart disease, unlike geriatric patients.[17] Additionally, there were no differences in the composition of oral antidiabetic drugs, rates of insulin use, length of insulin treatment, or total daily insulin dosages between individuals with DFUs who had low handgrip strength and those with normal grip strength.

Patients with normal grip strength used statins at a similar rate as patients with inferior grip strength. Statins were studied for their impact on patients' bodily functioning to assuage the worry that they would cause myopathy. However, the literature is contradictory. Numerous investigations.[28,29] could not find any evidence of a decline in functional performance or document any variation in muscular strength and capacity of exercise. The combination of statins plus exercise training, however, appeared to interact favorably with muscular response, performance, and proximal muscular strength, according to a number of studies.[28-30] According to a recent study, taking statins tempered the performance-enhancing effects of aerobic exercise.[31]

Furthermore, non-geriatric patients with diabetic foot disease exhibited higher rates of peripheral artery disease and diabetic neuropathy symptoms. These findings were consistent with those of an earlier study performed on geriatric patients.[17] Still, this study also revealed higher rates of diabetic retinopathy in non-geriatric patients with diabetic foot disease, unlike geriatric individuals. In individuals with peripheral artery disease, sarcopenia has been described as a predictive factor for overall survival.[32] Preclinical atherosclerosis, handgrip, and gait velocity were positively correlated in investigations.[33,34] Sarcopenia may be crucial in the early diagnosis of preclinical atherosclerosis, according to a recent study that found an association between the condition and the low ankle–brachial index, a measure used in clinical settings to identify peripheral arterial disease.[35] It is essential to conduct prospective studies in individuals with diabetic foot disorder to look at the substantial differences, particularly the impact of peripheral arterial disease on grip force and sarcopenia.

Because the study was record based, several patient-specific data, such as serum albumin and urine microalbumin levels, were absent. Furthermore, because this study is cross-sectional and observational, it cannot be concluded that a low or ideal grip force value can predict the likelihood of developing diabetic foot disease. Prospective studies, including larger sample size, are required to fully comprehend the impact of sarcopenia and handgrip strength in developing DFUs. Additionally, the study's sample population is not a good proxy for the entire population. This study's participants had longer diabetes histories and more complications because the study was conducted in a tertiary diabetes care facility. In this study, selection bias might be a possibility. Motor neuropathy may lead to reduced grip force, and in this study, we cannot exclude the independent effect of diabetic polyneuropathy on handgrip test results since the documentation of neuropathic symptoms is not a reliable diagnostic parameter for diabetic neuropathy.

In conclusion, this study underscores the significant association between low grip force and diabetic foot disorder in non-geriatric type 2 diabetic patients, with diabetic foot disorder showing a substantial correlation with low handgrip strength. The findings align with previous research on geriatric individuals, supporting the relevance of muscle strength as a potential risk factor for foot complications across different age groups. Furthermore, the study identifies potential influencing factors, such as age, body weight, and ischemic heart disease, which may contribute to the observed handgrip strength differences. The relationship between inflammation, vascular complications, and sarcopenia further highlights the importance of considering muscle health in managing diabetes patients with diabetic foot disease. Nevertheless, the study's limitations, including its cross-sectional design, preclude establishing causal relationships, necessitating prospective studies with larger cohorts to validate these findings.

Additionally, the absence of certain patient-specific data and the potential for selection bias require cautious interpretation. Nonetheless, these findings emphasize the importance of assessing handgrip strength in diabetes care, aiding in early detection and improved management of diabetic foot disease, and possibly offering valuable insights into the overall health status of diabetes patients. Future research in this area holds promise for refining preventive strategies and enhancing the quality of life for individuals with type 2 diabetes.

Ethics Committee Approval: This study was approved by Ethics Committee of SANKO University (protocol 2020/317).

Informed Consent: Verbal and written informed consent was obtained from the patients who agreed to take part in the study.

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