Original Article

The effects of spasticity on glucose metabolism and soft tissue body composition in patients with spinal cord injury

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ABSTRACT

Objectives: The aim of this study was to assess the effects of spasticity on glucose metabolism and percentage of fat-free mass (FFM%) in patients with spinal cord injury (SCI).

Patients and methods: A total of 33 patients (22 males, 11 females; mean age: 38.6±12.5 years; range, 20 to 64 years) with SCI defined by the American Spinal Injury Association Impairment Scale Grades A to D were included between September 2014 and May 2018. We assessed spasticity with the Modified Ashworth Scale (MAS) and evaluated spasms with the Penn Spasm Frequency Scale (PSFS). We assessed the glucose metabolism by calculating the Matsuda and HOMA-IR index, and measured FFM% by dual-energy X-ray absorptiometry.

Results: Fourteen patients had motor complete, and 19 had motor incomplete SCI. The neurological injury levels of the patients were C4-T12. There was a positive correlation between hip adductor muscle MAS and trunk, android, and gynoid FFM% and between hip extensor muscle MAS and android FFM% in patients with motor complete SCI. Hip extensor and knee flexor muscle MAS showed a negative correlation with the HOMA-IR. Hip adductor and extensor muscle MAS, as well as knee flexor and extensor muscle MAS, had a positive correlation with the Matsuda index in these patients. There was a positive correlation between knee extensor muscle MAS and gynoid FFM% and between PSFS and arms, trunk, gynoid, and total FFM% in patients with motor incomplete SCI. There was a negative correlation between hip adductor and extensor muscle MAS, PSFS, and level of fasting glucose in these patients.

Conclusion: This study supports the notion that spasticity has positive effects on the FFM% and glucose metabolism in patients with motor complete and incomplete SCI.

Keywords: Body composition, glucose, spasticity, spinal cord injury.

Diminished physical activity may cause muscle atrophy in patients with spinal cord injury (SCI). These patients may also show alterations in glucose metabolism and body composition.[1] Significant and rapid muscle atrophy below the injury level may occur in a short time such as within six weeks after an injury in patients with motor complete or incomplete SCI.[1-3] There is an increase in intramuscular fat deposition along with atrophy.[4] Elder et al.[5] showed that patients with motor complete SCI had a higher intramuscular fat amount and lower skeletal cross-sectional area, compared to the control group. They also observed that there was a correlation between intramuscular fat amount and glucose and insulin levels in the oral glucose test, suggesting that intramuscular fat amount might be a predictor of impaired glucose metabolism

and risk of type II diabetes mellitus (DM) in patients with SCI. Therefore, glucose metabolism may be impaired due to the increase in the intramuscular fat amount and alterations in the body composition of patients with SCI.

Researchers have shown that spasticity can preserve the muscle mass through muscle contraction in patients with SCI.[6,7] A few studies have proposed that spasticity may affect glucose metabolism positively, as it maintains fat-free mass (FFM) and prevents muscle atrophy in patients with only motor complete SCI.[8-10] In the present study, we aimed to investigate the impact of spasticity on glucose metabolism and FFM in both patients with motor complete SCI and patients with motor incomplete SCI.

PATIENTS AND METHODS

This single-center, prospective, cross-sectional study was conducted at the inpatient rehabilitation unit of Fatih Sultan Mehmet Training and Research Hospital between September 2014 and May 2018. Patients with SCI who were at least one-year post-injury were included in the study. Patients were not using any drugs that affect spasticity other than baclofen and the baclofen doses remained stable within the past six months. None of the patients were administered botulinum injections within the past six months. Patients having decubitus ulcers, urinary tract infections, and any other complications that affect spasticity, joint contracture, history of other central nervous system disease, an established diagnosis of DM, fasting glucose over 125 mg/dL, body mass index (BMI) higher than 30 kg/m², and those taking medications known to affect glucose metabolism such as glucocorticoids and others were excluded from the study. A total of 48 patients with SCI were screened. However, 11 patients were excluded, as the time span after the injury was shorter than one year, three patients due to DM, and one patient due to Parkinson's disease. Finally, the remaining 33 patients (22 males, 11 females; mean age: 38.6±12.5 years; range, 20 to 64 years) were included in the study (Figure 1). The patients were divided into two groups according to the American Spinal Injury Association (ASIA) Impairment Scale (AIS) A, B (motor complete) and AIS C, D (motor incomplete). A written informed consent was obtained from each patient. The study protocol was approved by the Fatih Sultan Mehmet Training and Research Hospital Ethics Committee (No: 2013/22). The study was conducted in accordance with the principles of the Declaration of Helsinki. The study is registered on ClinicalTrials.gov (NCT03859960).

Anthropometric measurements and body mass index

Waist and hip measurements were performed in each patient. The measurements were conducted in accordance with the recommendations of the World Health Organization (WHO). Patients were lying in bed in a supine, and vertical position to the bed. An inelastic tape was used without any compression. Waist circumference was measured from the midpoint between the top of the iliac crest to the last palpable rib at the end of expiration. Hip circumference was measured from the largest portion of the buttocks.[11] Wheelchair scale (Desis, BW, TUR) was used to measure the weight, when the patient was sitting on the wheelchair. The weight of the wheelchair
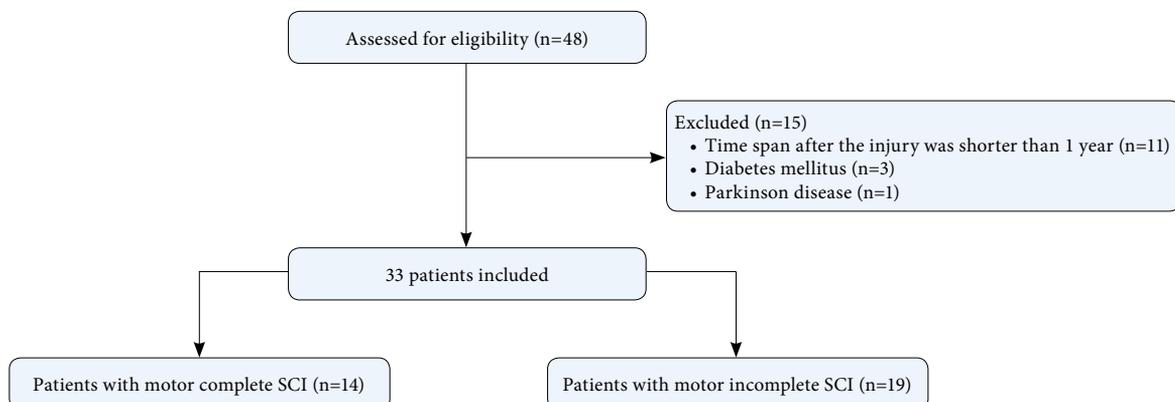

Figure 1. Study flow chart.
SCI: Spinal cord injury.

was, then, subtracted from the measurement. Thus, the weight of the patient was obtained. The BMI was calculated using the following formula: (body weight)/(square of height).

Body composition

A dual-energy X-ray absorptiometry (DXA) device (Lunar HVPS7681; General Electric, Madison, WI, USA) was used to measure the body composition as FFM% of arms, legs, android, gynoid, trunk, and total body.[12]

Assessment of spasticity

The Modified Ashworth Scale (MAS) and the Penn Spasm Frequency Scale (PSFS) were used to assess spasticity and spasm frequency, respectively. The lower extremity MAS scores of all patients were collected three times a day by a single researcher, owing to the changes in spasticity during the daytime. Hip extensor and adductor spasticity, knee extensor and flexor spasticity, and ankle plantar flexor spasticity were evaluated using the MAS in the first day at 10:00 A.M., 03:00 P.M., and 08:00 P.M. More importantly, we changed MAS 1 + Grade to 2, while MAS Grades 2, 3, and 4 were converted to 3, 4, and 5, respectively.[8,9,13] The patients were evaluated in the supine position, upper extremities parallel to the trunk, and the lower extremities parallel to each other, except for the evaluation of knee extensors in the prone position.[13] To determine the spasticity of each compartment, the average of the six measurements (three from the right and three from the left) was calculated.

Hip adductor spasticity MAS = (right hip adductor MAS 1 + right hip adductor MAS 2 + right hip adductor MAS 3 + left hip adductor MAS 1 + left hip adductor MAS 2 + left hip adductor MAS 3)/6. The average of spasticity was calculated for each compartment.

Flexor and extensor spasms were assessed using the PSFS developed by Penn et al.[14] It is a five-point subjective, self-report scale used to evaluate the spasm frequency.[15]

Glucose metabolic profiles

A total of 75 g oral glucose tolerance test (OGTT) was performed to all patients after 12-h overnight fasting. The patients' blood samples in the quantity of 2 mL were taken at 0, 30, 60, 90, and 120 min after loading the glucose solution. The plasma glucose levels were determined by the photometric method by using an Abbott ARCHITECT™ C16000 model auto-analyzer (Abbott Laboratories, Abbott Park, IL, USA). Insulin measurements were performed by the chemiluminescent micro-particle immunoassay method using the ARCHITECT™ system brand kits on the Abbott brand i2000SR immunological analyzer (Abbott Laboratories, Abbott Park, IL, USA). The HbA1c levels were measured by the ion exchange chromatography method using an automatic glycohemoglobin analyzer (ADAMS A1c HA-8180V; Arkray Inc., MN, USA).

TABLE 1						
Demographics and clinical characteristics of patients with SCI						
	Patients with motor complete SCI (n=14)			Patients with motor incomplete SCI (n=19)		
	n	Mean±SD	Median	n	Mean±SD	Median	*p**
Age (year)		37.4±9.9			39.6±14.3		0.601
Sex							0.136
Female	7			4			
Male	7			15			
Level of injury							0.278
Cervical	3			8			
Thoracic	11			11			
Time since injury (month)		47.4±52.6	23		62.1±47.8	55	0.183
Baclofen dose (mg/day)		22.9±22.3	25		29.5±34.7	0	0.803
BMI (kg/m²)		25.1±3.8			25.4±3.7		0.782
Waist circumference (cm)		91.3±11.5			94.6±11.6		0.419
Hip circumference (cm)		100.1±9.0			104.9±9.3		0.148
SCI: Spinal cord injury; BMI: Body mass index, * p<0.05.							

TABLE 2
Correlation between spasticity and FFM% in patients with motor complete and motor incomplete SCI

	Patients with motor complete SCI						Patients with motor incomplete SCI					
	Arms FFM%	Legs FFM%	Trunk FFM%	Android FFM%	Gynoid FFM%	Total FFM%	Arms FFM%	Legs FFM%	Trunk FFM%	Android FFM	Gynoid FFM%	Total FFM%
	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)
MAS hip adductor muscles	NS	NS	0.586 (0.035)*	0.604 (0.029)*	0.592 (0.033)*		NS	NS	NS	NS	NS	NS
MAS hip extensor muscles	NS	NS	NS	0.746 (0.003)*	NS	NS	NS	NS	NS	NS	NS	NS
MAS knee flexor muscles	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
MAS knee extensor muscles	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.529 (0.024)*	NS
MAS ankle plantar flexor muscles	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
PSFS	NS	NS	NS	NS	NS	NS	0.523 (0.026)*	NS	0.506 (0.032)*	NS	0.521 (0.027)*	0.526 (0.025)*

FFM%: Percentage of fat-free mass; SCI: Spinal cord injury; NS: Nonsignificant correlation; MAS: Modified Ashworth Scale; PSFS: Penn Spasm Frequency Scale; * p<0.05.

The Matsuda index and Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) were used to assess the glucose metabolism. The latter is a simple, widely accepted, and inexpensive method used for the assessment of insulin resistance. Although there is no absolute threshold value for HOMA-IR, most of the studies have accepted the values more than 2.7 for insulin resistance.[16] In this study, HOMA-IR was calculated using the following formula: fasting insulin (µIU/mL) × fasting plasma glucose (mg/dL)/405.[17] Insulin sensitivity of the patients was calculated using the Matsuda index formula: 10.000/square root (fasting plasma insulin × fasting plasma glucose) × (mean OGTT insulin concentration × mean OGTT glucose concentration).[18]

Statistical analysis

The power analysis was calculated according to the study by Gorgey et al.[9] who assumed knee flexor spasticity with effect size as 0.730 and power as 0.80 (alpha=0.05). The sample sizes of minimum 12 patients were calculated for both the motor complete and motor incomplete groups. This calculation was performed by G*Power version 3.0.10 software (Heinrich-Heine-Universität Düsseldorf, Düsseldorf, Germany).
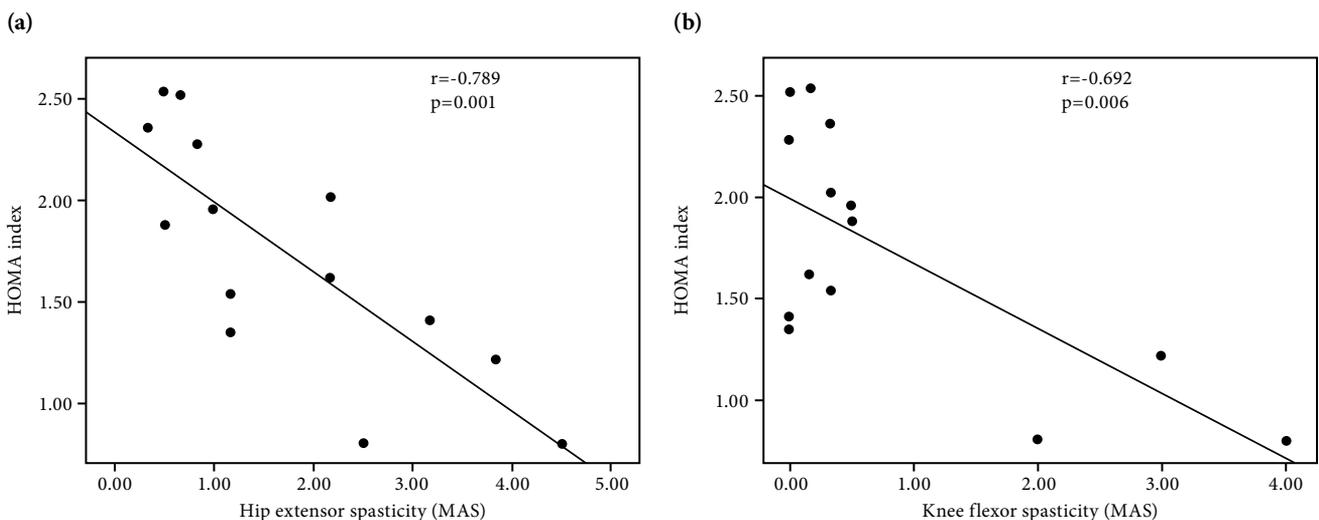
(a)

(b)

Figure 2. Correlation between spasticity and HOMA index in patients with motor complete SCI. **(a)** Correlation between hip extensor muscle MAS and HOMA index; **(b)** Correlation between knee flexor muscle MAS and HOMA index.
HOMA: Homeostasis Model Assessment; SCI: Spinal cord injury; MAS: Modified Ashworth Scale.

Statistical analysis was performed using the IBM SPSS version 22.0 software (IBM Corp., Armonk, NY, YSA). The Shapir-Wilk test was used to check whether the variables were normally distributed. Descriptive data were expressed in mean ± standard deviation (SD), median (min-max) or number and frequency. The Student's t-test was used to compare normally distributed variables between the two groups. Additionally, the two groups of variables with non-normal distribution were compared using the Mann-Whitney U test. Qualitative data were compared using the Fisher's exact test. Pearson correlation analysis was performed to assess the association between the variables with conformity to the normal distribution. Where the baclofen dose was fixed, partial correlation was used to evaluate the associations. A p value of <0.05 was considered statistically significant.

RESULTS

Fourteen patients had motor complete (AIS A, B) and 19 patients had motor incomplete (AIS C, D) SCI. The neurological injury levels of patients were C4-T12. The time from the event varied between 12 and 190 months with a mean time of 55.6±49.6 months. There was no statistically significant difference between the two groups in terms of sex, age, time from event, baclofen dose, BMI, and waist and hip circumferences ($p>0.05$) (Table 1). Additionally, there was no significant difference between the two groups in terms of fasting
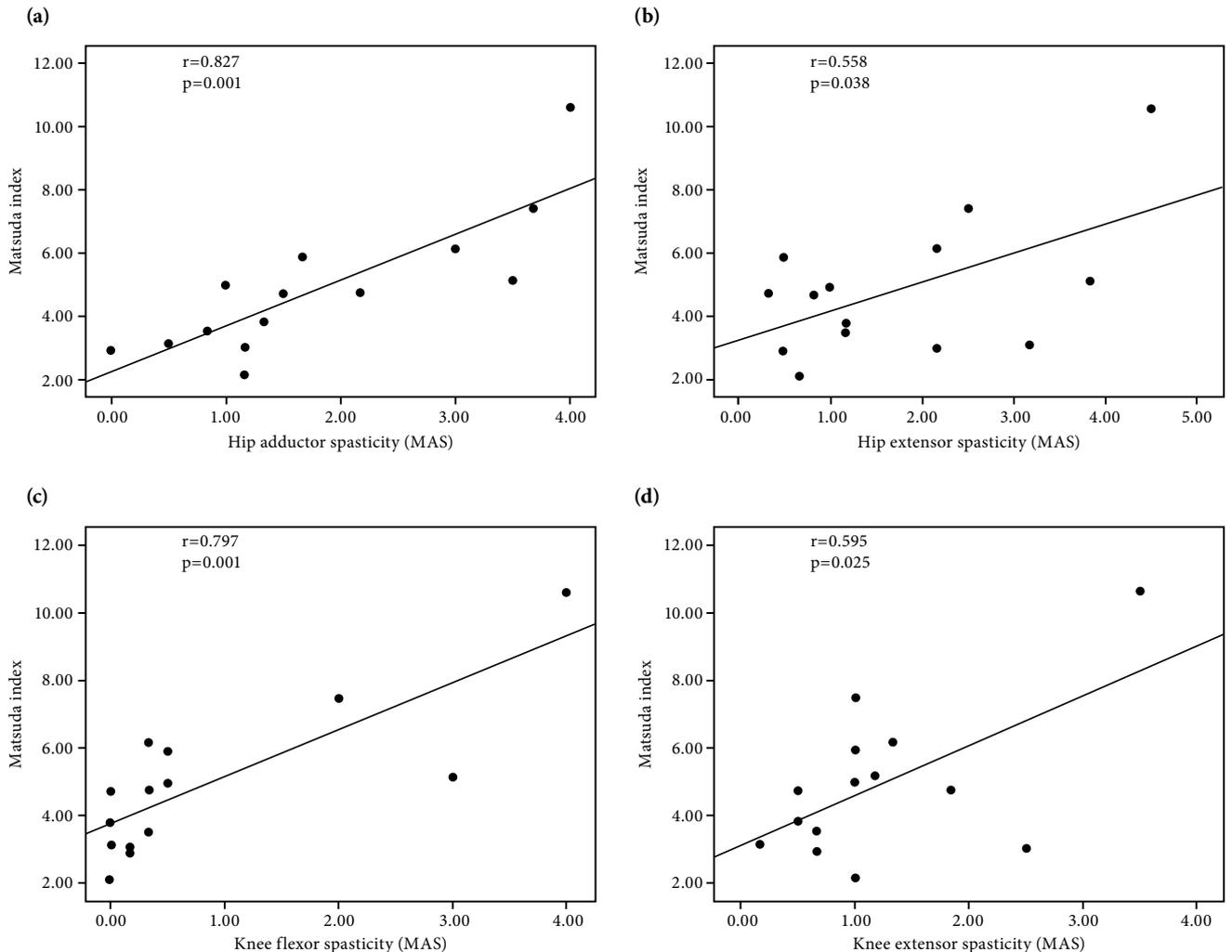

Figure 3. Correlation between spasticity and Matsuda index in patients with motor complete SCI. **(a)** correlation between hip adductor muscle MAS and Matsuda index; **(b)** correlation between hip extensor muscle MAS and Matsuda index; **(c)** correlation between knee flexor muscle MAS and Matsuda index; **(d)** correlation between knee extensor muscle MAS and Matsuda index.

SCI: Spinal cord injury; MAS: Modified Ashworth Scale.

TABLE 3
Correlation between spasticity and fasting glucose levels, HbA1c levels, HOMA-IR, and Matsuda index in patients with motor complete and motor incomplete SCI

	Patients with motor complete SCI				Patients with motor incomplete SCI			
	Fasting glucose level	HbA1c	HOMA-IR	Matsuda index	Fasting glucose level	HbA1c	HOMA-IR	Matsuda index
	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)
MAS hip adductor muscles	NS	NS	NS	0.827 (0.001)*	-0.607 (0.006)*	NS	NS	NS
MAS hip extensor muscles	NS	NS	-0.789 (0.001)*	0.558 (0.038)*	-0.505 (0.027)*	NS	NS	NS
MAS knee flexor muscles	NS	NS	-0.692 (0.006)*	0.797 (0.001)*	NS	NS	NS	NS
MAS knee extensor muscles	NS	NS	NS	0.595 (0.025)*	NS	NS	NS	NS
MAS ankle plantar flexor muscles	NS	NS	NS	NS	NS	NS	NS	NS
PSFS	NS	NS	NS	NS	-0.437 (0.041)*	NS	NS	NS

HOMA-IR: Homeostasis Model Assessment of Insulin Resistance; SCI: Spinal cord injury; NS: Nonsignificant correlation; MAS: Modified Ashworth Scale; PSFS: Penn Spasm Frequency Scale; * p<0.05.

glucose levels, HbA1c, HOMA-IR, Matsuda index, hip extensor and adductor muscle MAS, knee flexor and extensor muscle MAS, ankle plantar flexor muscle MAS, PSFS and FFM% (p>0.05).

In the patients with motor complete SCI, the waist circumference had a negative correlation with the hip extensor muscle MAS and ankle plantar flexor muscle MAS (r=-0.547, p=0.043; r=-0.613, p=0.020, respectively). Additionally, the waist circumference had a positive correlation with ankle plantar flexor muscle MAS (r=0.47, p=0.042) in the patients with motor incomplete SCI. However, there was no statistically significant correlation between the hip circumference and other variables in either group.

TABLE 4
Partial correlation between spasticity and fasting glucose level, HbA1c, HOMA-IR, and Matsuda index where baclofen dose was fixed

	Patients with motor complete SCI				Patients with motor incomplete SCI			
	Fasting glucose level	HbA1c	HOMA-IR	Matsuda index	Fasting glucose level	HbA1c	HOMA-IR	Matsuda index
	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)	r (p)
MAS hip adductor muscles	NS	NS	NS	0.784 (0.002)*	-0.587 (0.010)*	NS	NS	NS
MAS hip extensor muscles	NS	NS	-0.748 (0.003)*	NS	-0.469 (0.049)*	NS	NS	NS
MAS knee flexor muscles	NS	NS	-0.617 (0.025)*	0.710 (0.007)*	NS	NS	NS	NS
MAS knee extensor muscles	NS	NS	NS	NS	NS	NS	NS	NS
MAS ankle plantar flexor muscles	NS	NS	NS	NS	NS	NS	NS	NS
PSFS	NS	NS	NS	NS	-0.476 (0.046)*	NS	NS	NS

HOMA-IR: Homeostasis Model Assessment of Insulin Resistance; SCI: Spinal cord injury; NS: Nonsignificant correlation; MAS: Modified Ashworth Scale; PSFS: Penn Spasm Frequency Scale; * p<0.05.

The hip adductor muscle MAS showed a positive correlation with FFM% android, FFM% gynoid, and FFM% trunk proportions in the patients with motor complete SCI. The hip extensor muscle MAS had also a positive correlation with android FFM% proportion (p<0.05). However, the PSFS scores were not associated with FFM% (p>0.05) (Table 2).

We observed that the knee extensor muscle MAS had a positive correlation with gynoid FFM% in the patients with motor incomplete SCI. The PSFS had a positive correlation with arms FFM%, trunk FFM%, gynoid FFM%, and total body FFM% proportions (p<0.05) (Table 2).

The hip extensor muscle MAS and knee flexor muscle MAS had a negative correlation with HOMA-IR in the patients with motor complete SCI (p<0.05) (Figure 2). The hip adductor and extensor muscle MAS, as well as knee flexor and extensor muscle MAS had a positive correlation with the Matsuda index (p<0.05) (Figure 3). Any type of muscle MAS had no correlation with fasting plasma glucose and HbA1c levels. Additionally, the PSFS scores had no correlation with the HOMA-IR, Matsuda index, fasting plasma glucose, and HbA1c levels (p>0.05) (Table 3).

The hip, knee, and ankle muscle MAS showed no correlation with the HbA1c, HOMA-IR, and Matsuda index in the patients with motor incomplete SCI (p>0.05). However, the fasting plasma glucose level had a negative correlation with the hip adductor and extensor muscle MAS and PSFS (p<0.05) (Table 3).

The partial correlation analysis between spasticity and fasting glucose, HbA1c, HOMA-IR and Matsuda index, where baclofen dose were fixed, showed that the hip extensor muscle MAS and knee flexor muscle MAS had a negative correlation with HOMA-IR. Additionally, the hip adductor muscle MAS and knee flexor muscle MAS had a positive correlation with the Matsuda index in the patients with motor complete SCI. Fasting plasma glucose levels showed a negative correlation with hip adductor and hip extensor muscle MAS, and a partial correlation with PSFS in the patients with motor incomplete SCI (Table 4).

DISCUSSION

In the present study, we investigated the impact of spasticity on glucose metabolism and body composition in the patients with SCI. Our study results showed a significant correlation between spasticity and insulin resistance, as well as insulin sensitivity in the patients with motor complete SCI. We also found a significant correlation between spasticity and fasting plasma glucose levels in the patients with motor incomplete SCI. Spasticity was correlated with FFM% in the patients with both motor complete and motor incomplete SCI.

Glucose intolerance and DM are more common in patients with SCI than the general population.[19] This phenomenon is explained by inactivation, alteration in body composition, and an increase in the adipose tissue.[20] Hormonal alterations may also occur after SCI. A decrease in testosterone, growth hormone, and insulin growth factor is observed in SCI, thereby probably leading to a decline in FFM and rise in fat mass which may indirectly increase the risk of metabolic syndrome and heart disease, eventually.[1,21-22] Therefore, heart disease has been shown to be one of the most common reasons for death in these patients at least one year after injury.[23] It is of utmost importance that healthcare professionals should be aware of these changes.

In the current study, we found a positive correlation between the hip adductor muscle MAS and trunk, android, and gynoid FFM% rates and between the hip extensor muscle MAS and android FFM% in patients with motor complete SCI. These results are also consistent with the results of previous studies.[8,9] In a cross-sectional study, Gorgey et al.[9] found a positive correlation between knee extensor muscle MAS along with gynoid FFM% and total body FFM%; knee flexor spasticity along with trunk FFM% and total body FFM% in patients with chronic motor complete SCI. They also showed that spasticity might have an effect on glucose metabolism via involuntary muscle contractions which decrease muscle atrophy, thereby maintaining FFM% and preventing increase in FM%. Furthermore, Jung et al.[8] reported that fat mass was lower in patients with chronic motor complete SCI with severe spasticity than in those with mild spasticity. They also found a negative correlation between the sum of the knee extensor and ankle extensor MAS and the percentage of body fat. In addition, we evaluated patients with motor incomplete SCI in our study and found a positive correlation between the knee extensor muscle MAS and gynoid FFM% rates and between PSFS and arms, gynoid, trunk and total body FFM% proportions in these patients.

The MAS, which is the most common scale used in this setting, was applied to assess spasticity in our study. While choosing the most optimal measurement

tool, it should be taken into account that spasticity is a multidimensional phenomenon.[24] The severity of spasticity can alter within a single day depending on the patient's physical and emotional conditions.[25] Therefore, patients were assessed three times a day, due to the fluctuating nature of spasticity. Unlike other studies, PSFS was also used in our study, since spasms cause involuntary muscle contractions in patients with SCI. The PSFS is a self-report measure which assesses spasm frequency, while MAS is an examiner-based scale; therefore, we attempted to measure the multiple aspects of spasticity with a wider view.[15,26,27]

Furthermore, Gorgey et al.[9] found a correlation between spasticity and body composition, although they were unable to show any correlation between spasticity and insulin sensitivity and insulin resistance in patients with motor complete SCI. They suggested that spasticity might indirectly affect the glucose metabolism, by affecting the body composition. In our study, we found a negative correlation between the hip extensor muscle MAS and knee flexor muscle MAS with insulin resistance in the patients with motor complete SCI and a positive correlation between hip extensor, hip adductor, knee extensor and knee flexor muscle MAS with insulin sensitivity. These results indicate that spasticity may exert a positive effect on the glucose metabolism. In the assessment of partial correlation, where the baclofen dose was fixed in patients with motor complete SCI, we found that insulin resistance had a negative correlation with hip extensor muscle MAS and the knee flexor muscle MAS. Additionally, insulin sensitivity had a positive correlation with hip adductor muscle MAS and knee flexor muscle MAS. Jung et al.[8] assessed fasting plasma glucose in their study, but they did not assess the insulin sensitivity. They found significantly lower plasma glucose levels in patients with motor complete SCI who had severe spasticity, compared to those with mild spasticity.

Apart from spasticity, voluntary muscle contractions have an additional impact on the muscle metabolism in patients with motor incomplete SCI. Therefore, in these patients, it is difficult to discriminate the exclusive impact of spasticity on glucose metabolism and FFM%. Nevertheless, we suggest that spasticity and spasms may have a positive effect on the glucose metabolism and body composition, since fasting glucose levels had a negative correlation with hip adductor and extensor muscle MAS and PSFS in both Pearson's correlation analysis and baclofen dose-adjusted partial correlation

analysis in this study. Furthermore, spasticity and spasms were found to have a correlation with FFM% in the patients with motor incomplete SCI.

An experimental, controlled study including patients with SCI based on catheter measurements showed that the glucose uptake was three times more in the legs of patients with SCI, compared to the control group.[10] This finding suggests that spasticity may decrease the risk of DM in patients with SCI independently from insulin.

In the current study, we found a negative correlation between the waist circumference and hip extensor muscle MAS and ankle plantar flexor muscle MAS in the patients with motor complete SCI. However, the ankle plantar flexor muscle MAS and waist circumference were positively correlated in the patients with motor incomplete SCI. Waist circumference is a determinant for visceral adipose tissue and researchers have reported an association between visceral adipose tissue and lipid and carbohydrate metabolism in patients with SCI.[28] Moreover, the relationship between cardiovascular risk and waist circumference was shown in patients with SCI.[29] Jung et al.[8] found that the waist circumference was significantly lower in patients with motor complete SCI with severe spasticity, compared to the patients with motor complete SCI with mild spasticity. These findings are consistent with the results of the patients with motor complete SCI in this study. However, an inverse relationship was identified in the patients with motor incomplete SCI. Therefore, further studies are required to clarify this issue.

The inclusion of the patients with paraplegia and tetraplegia is the main limitation of this study. Since the physical activity levels of the patients with paraplegia and tetraplegia are different, these two patient groups should be separately analyzed. Another limitation of this study may be the lack of upper limb spasticity assessment in tetraplegic patients.

In conclusion, our study results suggest that spasticity may have a positive effect on glucose metabolism and FFM% in patients with both motor complete and motor incomplete SCI. In the practice of rehabilitation, the positive effects of spasticity on the metabolic profiles and FFM% in patients with chronic motor complete and incomplete SCI should be considered.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

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