Research Article

Body composition and metabolic parameters in men with chronic traumatic paraplegia – A pilot study from India

Kalyani Sridharan[1], Shipra Rachna Singh[2], Kripa Elizabeth Cherian [1], Nitin Kapoor[1], Jane Elizabeth[2], Judy Ann John[2], Nihal Thomas[1], Thomas V. Paul [1]

[1]Department of Endocrinology, Diabetes and Metabolism, Christian Medical College, Vellore, India, [2]Department of Physical Medicine and Rehabilitation, Christian Medical College, Vellore, India

Objective: To study body composition, measures of insulin resistance and dyslipidemia in Indian men with paraplegia as compared to age and body mass index (BMI) matched able-bodied men.
Design: Cross sectional study
Setting: Departments of Physical Medicine and Rehabilitation and Endocrinology
Participants: Males aged 18–45 years with chronic traumatic paraplegia versus age and BMI-matched able-bodied men
Interventions: None
Main outcome measures: Measures of body composition such as total body fat, lean mass, regional adiposity using dual energy x-ray absorptiometry (DXA), metabolic profile and insulin resistance
Results: Subjects with paraplegia ($n = 43$), compared to controls ($n = 36$), had higher %Fat mass (FM) (25.5 (21.2–28.9) vs 20.2 (15.9–22.2); $P < 0.01$), lower trunk to leg ratio (0.66 (0.51–0.73) vs 0.87 (0.72–0.94); $P < 0.01$), lower lean mass index (14.38 (2.57) vs 17.80 (2.34); $P < 0.01$) and lower appendicular lean mass index (5.81 ± 1.26 vs 8.17 ± 1.12; $P < 0.01$). Fasting blood glucose (mg/dl) was higher (89.0(81.5–96.5) vs 80.0 (74.5–88.2); $P < 0.01$), Homeostasis model assessment of insulin resistance was higher (1.33 (1.03–2.12) vs 0.94 (0.52–1.78); $P = 0.02$), Quantitative insulin sensitivity check index (QUICKI) was lower (0.36 ± 0.04 vs 0.38 ± 0.05; $P = 0.02$) and HDL-C was lower (33.00 (30.00–42.75) vs 38.50 (33.00–43.25); $P < 0.02$) in cases compared to controls. QUICKI correlated positively with HDL-C and negatively with %FM, estimated VAT volume and TG. Trunk to leg ratio correlated positively with TG even after controlling for %FM.
Conclusion: Men with chronic paraplegia had lower lean mass, higher total and regional fat mass, increased insulin resistance and low HDL-C when compared with BMI-matched able-bodied controls. Both total and regional adiposity correlated with poor metabolic profile.

Keywords: Paraplegia, Body composition, DXA, HOMA-IR, QUICKI

Introduction

Traumatic spinal cord injury (SCI) impacts predominantly young adults resulting in significant individual and societal costs. Prevalence of obesity increases after SCI with a consequent deterioration in metabolic parameters.[1] Body mass index (BMI), a widely used tool for measuring obesity, has been shown to underestimate the percent fat mass in subjects with SCI.[2] Some studies have recommended the use of lower BMI cutoffs to better define obesity in this population.[3,4] However, since there are no well-defined validated BMI cutoffs in subjects with SCI, and since BMI does not reflect changes in regional adiposity, body composition is a better marker of obesity and metabolic risk in this population.[5]

Measurement of body weight embodies both fat mass and fat-free mass (or for practical terms, lean body mass) and cannot assess the two components separately. However, assessment of body composition by

Correspondence to: Dr. Thomas V. Paul, Dept. of Endocrinology, Diabetes and Metabolism, Christian Medical College, Vellore (TN) - 632004, India; Ph: +91- 9566920379(M). Email: thomasvpaul@yahoo.com

DOI 10.1080/10790268.2021.1888021

dual-energy x-ray absorptiometry (DXA) allows for measurement of amount and distribution of the two components.

The immediate and profound skeletal muscle atrophy that follows SCI results in a decrease in lean mass below the level of injury.[6] Studies have demonstrated lower fat-free mass and higher fat mass post SCI in comparison to able-bodied individuals in spite of similar BMIs.[7] This decline in lean mass correlates inversely with age and duration of injury and depends on the level and completeness of injury.[8] A more complete and a higher level of injury causes a greater decline in lean mass. Subjects with tetraplegia have a reduced lean mass in the upper limbs compared to those with paraplegia.[5] The loss of metabolically active muscle mass combined with physical inactivity due to the disability decreases the basal metabolic rate and total energy expenditure resulting in obesity.[9]

Studies demonstrate an increase not only in whole-body fat mass but also in regional fat mass.[8] Trunk and leg fat mass were found to be greater in subjects with SCI when compared to controls.[10] Measures of central adiposity like visceral adipose tissue mass was found to be higher in subjects with SCI as compared to able-bodied individuals even after controlling for waist circumference.[11]

The decrease in lean mass and increase in fat mass predisposes individuals with SCI to dysglycemia, dyslipidemia and cardiovascular disease.[12,13] The loss in muscle mass was found to be associated with reduced whole-body insulin-mediated glucose utilization.[14] The increase in whole body and regional adiposity has been shown to correlate positively with liver adiposity, insulin resistance and triglyceride levels and inversely with high-density lipoprotein cholesterol (HDL-C) levels.[15,16] While many studies support the above associations, a systematic review by Wilt *et al.* did not suggest an increased metabolic risk in subjects with SCI.[17]

Ethnic differences in BMI and its correlation with body fat in able-bodied individuals are widely acknowledged.[18–20] In a series of comparative studies, Deurenberg *et al.* demonstrated that Asians have a higher percentage of body fat at a given BMI compared to Caucasians.[21,22] Rush EC *et al.* demonstrated that Asian Indians have more fat, altered fat distribution (predominantly abdominal) with less lean mass and bone mineral content compared to Europeans and Polynesians.[19] These differences in body composition contribute to increased insulin resistance and greater prevalence of metabolic syndrome among Asians particularly Indians.[18] These ethnic differences in body composition are believed to be due to differences in genes, intra-uterine environment and physical activity and dietary patterns.[20] High protein diet and exercise program has been shown to improve insulin resistance in individuals post SCI.[23]

In view of these differences in body composition in able-bodied individuals between Caucasians and Indians, we hypothesized that the changes induced by paraplegia in Indians may not mirror that seen in the Caucasians. Only two Indian studies exist which looked at body composition or metabolic parameters in subjects with SCI. Tharion *et al.*[24] studied 48 subjects with chronic SCI and found them to have low HDL-C and high blood glucose while Singh R *et al.*[25] studied subjects with acute SCI and found a decrease in lean mass and increase in adiposity during the first year post injury. However, none of these studies had a control group. The aim of the present study is to examine body composition, measures of insulin resistance and lipid profile in paraplegic men one to three years following a traumatic spinal cord injury and to compare these parameters with age, and BMI-matched able-bodied men. We also tried to examine the correlation between body composition and metabolic indices.

Methods

Subjects

The study was a cross sectional observational study done at a tertiary care center in Southern India. Subjects with chronic traumatic paraplegia who attended the department of Physical Medicine and Rehabilitation (PMR) were recruited from April 2016 to May 2019 into the study. Consecutive men aged 18–45 years with traumatic paraplegia between 1 and 3 years post injury were recruited. All men had motor complete injury with neurological level between T2 and L1 and belonged to American Spinal Cord Injury Association (ASIA) impairment scale A or B.[26] Patients on drugs which are known to affect bone mineral metabolism, those with renal failure, severe infections, history of malabsorption, patients on treatment for osteoporosis like calcium, vitamin D and bisphosphonates, patients who have psychiatric illness or not capable or willing to give informed consent and patients in whom DXA scan is not possible due to deformities were excluded from the study. Age, and BMI-matched able-bodied men were taken as controls. They were recruited from the local community network. All study subjects signed Informed consent.

Clinical examination and anthropometric measures

The study was approved by the institutional review board. Patients who gave informed consent and fulfilled the inclusion criteria were recruited for the study. Subjects with paraplegia were classified into ASIA grade A or B. ASIA grade A were those with no sensory or motor function preserved in the sacral segments S4–S5 while ASIA grade B were those with sensory but no motor function below the level of injury (including sacral segments S4–S5). Modified Ashworth Scale (MAS)[27] was used to grade the level of spasticity in these subjects based on which they were divided into MAS category 1 who had a score less than 2 (nil to slight increase in muscle spasticity in the paralyzed lower limbs) and MAS category 2 who had a score greater than or equal to 2 (more marked to severe muscle spasticity in the paralyzed lower limbs). Bodyweight was measured using an electronic scale (range 400 g to 200 kg) to the nearest 0.1 kg. Since the majority of patients could not ambulate, recumbent length was measured to the nearest 0.1 cm. BMI was subsequently calculated as weight/length^2 (kg/m^2). Waist circumference was measured midway between lower costal margin and anterior superior iliac spine with the patient in supine position using a stretch-resistant tape.

Assessment of body composition

Body composition was assessed by DXA scan (Hologic QDR 4500 Discovery A; Hologic, Bedford, MA). The scans were performed by a single technician and the coefficient of variance (%CV) for the measures of body composition were <3%. Parameters of body composition that were analyzed included percent total body fat (%FM), fat mass index (FMI) (fat mass/height^2) (kg/m^2), percent trunk fat to percent leg fat ratio (% trunk fat/% leg fat), estimated visceral adipose tissue volume (VAT volume) (cm^3), lean mass index (LMI) (lean mass/height^2)(kg/m^2) and appendicular lean mass index (ALMI) (appendicular lean mass/height^2) (kg/m^2).

Biochemical parameters

Samples were collected after a 12 h overnight fast for plasma glucose, serum insulin and serum lipid profile. Fasting plasma glucose was measured by hexokinase method using an automated glucose analyzer and serum insulin was measured by chemiluminiscent immunoassay. Homeostatic model assessment of insulin resistance (HOMA-IR) and Quantitative insulin sensitivity check index (QUICKI) was calculated from the above two parameters as per the formulas given by Matthews *et al.*[28] and Katz *et al.*,[29] respectively. Total cholesterol (TC), High-density lipoprotein cholesterol (HDL-C) and triglycerides (TG) were estimated by colorimetric method using an automated chemistry analyzer and low-density lipoprotein cholesterol (LDL-C) was calculated using Friedwald's formula LDL-C = TC – HDL-C – (TG/5).[30]

Statistical analysis

Continuous variables were expressed as mean and standard deviation or median and interquartile range (IQR) while categorical variables were expressed as ratios or percentages. Independent *t*-test and Mann–Whitney U test were used to compare parametric data and non-parametric data, respectively. Chi-square test was used for categorical variables. Pearson's correlation was performed between measures of body composition and metabolic indices. A *post hoc* subgroup analysis by MAS category was done on the outcome measures of body composition and insulin resistance. A P value of less than 0.05 was taken as significant. Statistical analysis was performed by Statistical Package of Social Sciences (SPSS) version 25.0.

Results

Baseline characteristics

A total of 43 men with chronic traumatic paraplegia and 36 age, and BMI-matched able-bodied men were

Table 1 Baseline characteristics of subjects with paraplegia and able bodied controls.

Variable	Paraplegia (*N* = 43)	Able bodied controls (*N* = 36)	P value
Age (years)	30.91 (6.5)	32.36 (4.9)	0.27
BMI (kg/m^2)	21.2 (4.4)	22.7 (3.3)	0.08
Waist circumference (cm)	84.50 (70.75–90.00)	92.50 (88.00 –96.00)	<0.01
Time since injury (months)	27 (18–39)	–	–
Time since ambulation (months)	6.5 (2–12.5)	–	–
ASIA category, n/N	36/43	–	–
A B	7/43		
Neurological level, n/N		–	–
Thoracic T3-T9	17/43		
Thoracic T10-T12	24/43		
Lumbar	2/43		
Modified Ashworth score (MAS) Category, n/N		–	–
1 (MAS score <2)	34/43		
2 (MAS score ≥ 2)	9/43		

Note: Values in Mean (SD) or Median (IQR). ASIA – American Spinal cord Injury Association Impairment Scale.

recruited for the study. The baseline characteristics of the study and control subjects are depicted in Table 1. The majority of subjects with paraplegia belonged to ASIA category A, MAS category 1 and had a thoracic spinal level of injury. Age and BMI were similar between the groups but subjects with paraplegia had a lower waist circumference (84.50 cm (70.75 cm – 90 cm)) compared to able-bodied controls 92.50 cm (88 cm – 96 cm)($P < 0.01$). 72% of the patients were wheelchair bound while the remaining could ambulate with the help of Knee Ankle Foot Orthrosis (KAFO) plus either elbow crutches or walker.

Body composition parameters between subjects with paraplegia and able-bodied controls

%FM though not FMI was significantly greater in subjects with paraplegia (25.5 (21.2–28.9)) vs (20.2 (15.9–22.2); $P < 0.01$). Regional adiposity was also increased as evidenced by lower %trunk/%leg fat as compared to able-bodied controls (0.66 (0.51–0.73) vs 0.87 (0.72–0.94); $P < 0.01$). Estimated VAT volume (cm^3) was not significantly different between the two groups (370 (259–428) vs 320(255–399); $P = 0.33$). LMI and ALMI was lower in subjects with paraplegia compared to controls (LMI (kg/m^2): 14.38 (2.57) vs 17.80 (2.34); $P < 0.01$; ALMI (kg/m^2): 5.81 (1.26) vs 8.17 (1.12); $P < 0.01$) (Table 2).

Metabolic indices between subjects with paraplegia and able-bodied controls

Fasting blood glucose, measures of insulin resistance and lipid profile were assessed in 33 subjects with paraplegia and 34 able-bodied controls. Median fasting blood glucose (mg/dl) were significantly higher in subjects with paraplegia 89.0 (81.5–96.5) vs 80.0 ((74.5–88.2); $P < 0.01$). HOMA-IR was significantly higher (1.33 (1.03–2.12) vs 0.94 (0.52–1.78); $P = 0.02$) while QUICKI was lower in subjects with paraplegia as compared to controls (0.36 (0.04) vs 0.38 (0.05); $P = 0.02$). Lipid profile was similar between the two groups except for a significantly lower HDL Cholesterol (mg/dl) in the paraplegia group (33.00(30.00–42.75) vs 38.50(33.00–43.25); $P = 0.04$) (Table 3).

Subgroup analysis of measures of body composition and metabolic indices by MAS category LMI and ALMI were significantly higher in the subgroup with greater muscle spasticity (MAS category 2) (LMI (kg/m^2): 16.07 (1.86) vs 13.93 (2.56); $P = 0.01$; ALMI (kg/m^2): 6.69 (1.11) vs 5.58 (1.20); $P = 0.02$). Measures of adiposity and metabolic indices did not differ between the two groups (Table 4).

Correlation between measures of body composition and metabolic indices

QUICKI correlated negatively with measures of adiposity like %FM (Pearson's $r = -0.38$; $P < 0.01$), FMI ($r = -0.30$; $P = 0.02$) and estimated VAT volume ($r = -0.26$; $P = 0.04$) but not with %trunk/% leg fat. There was a significant positive correlation of QUICKI with HDL-Cholesterol ($r = 0.26$; $P = 0.05$) and negative correlation with TG ($r = -0.35$; $P < 0.01$) (Figure 1). No significant correlation between QUICKI and LMI or ALMI was found (PFIG = 0.26).

Measures of regional adiposity like %trunk/%leg fat correlated positively with ALMI ($r = 0.63$; $P < 0.01$) and TG ($r = 0.37$; $P < 0.01$) (Figure 2). On partial correlations, the correlation between %trunk/%leg fat with

Table 2 Body composition parameters between subjects with paraplegia and able bodied controls.

Variable	Paraplegia ($n = 43$)	Able bodied controls ($n = 36$)	*P* value
Lean mass index (kg/m^2)	14.38 (2.57)	17.80 (2.34)	<0.01
Appendicular lean mass index (kg/m^2)	5.81 (1.26)	8.17 (1.12)	<0.01
Total body fat %	25.5 (21.2–28.9)	20.2 (15.9–22.2)	<0.01
Fat mass/ height^2 (kg/m^2)	5.18 (3.47–6.59)	4.95 (3.06–5.66)	0.08
%Trunk fat/%leg fat	0.66 (0.51–0.73)	0.87 (0.72–0.94)	<0.01
Estimated visceral adipose tissue volume (cm^3)	370 (259–428)	320 (255–399)	0.33

Note: Values in Mean (SD) or Median (IQR).

Table 3 Metabolic indices between subjects with paraplegia and able bodied controls.

Variable	Paraplegia ($n = 33$)	Able bodied Controls ($n = 34$)	P value
Fasting blood glucose (mg/dl)	89.0 (81.5–96.5)	80.0 (74.5–88.2)	<0.01
Fasting Insulin (mU/dl)	5.90 (4.65–9.10)	4.75 (2.60–8.15)	0.20
HOMA-IR	1.33 (1.03–2.12)	0.94 (0.52–1.78)	0.02
QUICKI	0.36 (0.04)	0.38 (0.05)	0.02
Total cholesterol (mg/dl)	164.12 (44.0)	170.05 (36.35)	0.55
Triglycerides (mg/dl)	99.50 (82.50–165.50)	110.00 (72.75–158.00)	0.63
HDL-Cholesterol (mg/dl)	33.00 (30.00–42.75)	38.50 (33.00–43.25)	0.04
LDL-Cholesterol (mg/dl)	109.90 (33.88)	112.88 (31.04)	0.71

Note: Values in Mean (SD) or Median (IQR). HOMA-IR – Homeostatic model assessment of insulin resistance; QUICKI – Quantitative insulin sensitivity check index.

Table 4 Subgroup analysis of measures of body composition and metabolic indices by Modified Ashworth Score (MAS) category.

Variable	MAS Category 1 (n = 34)	MAS Category 2 (n = 9)	P value
Lean Mass index (kg/m^2)	13.93 (2.56)	16.07 (1.86)	0.01
Appendicular lean mass index (kg/m^2)	5.58 (1.2)	6.69 (1.11)	0.02
% Total body fat	24.70 (21.12–28.20)	28.90 (21.65–30.70)	0.28
%Trunk fat/%leg fat	0.63 (0.50–0.71)	0.73 (0.60–0.80)	0.13
QUICKI	0.35 (0.03)	0.36 (0.02)	0.60
HOMAIR	1.33 (1.06–2.14)	1.31 (0.99–2.34)	0.57
HDL-Cholesterol (mg/dl)	33.00 (30.00–43.50)	33.00 (29.00–35.00)	0.47
Triglyceride (mg/dl)	98.00 (83.00–154.50)	135.00 (81.00–197.00)	0.50

Note: Values in Mean (SD) or Median (IQR). HOMA-IR – Homeostatic model assessment of insulin resistance; QUICKI – Quantitative insulin sensitivity check index.

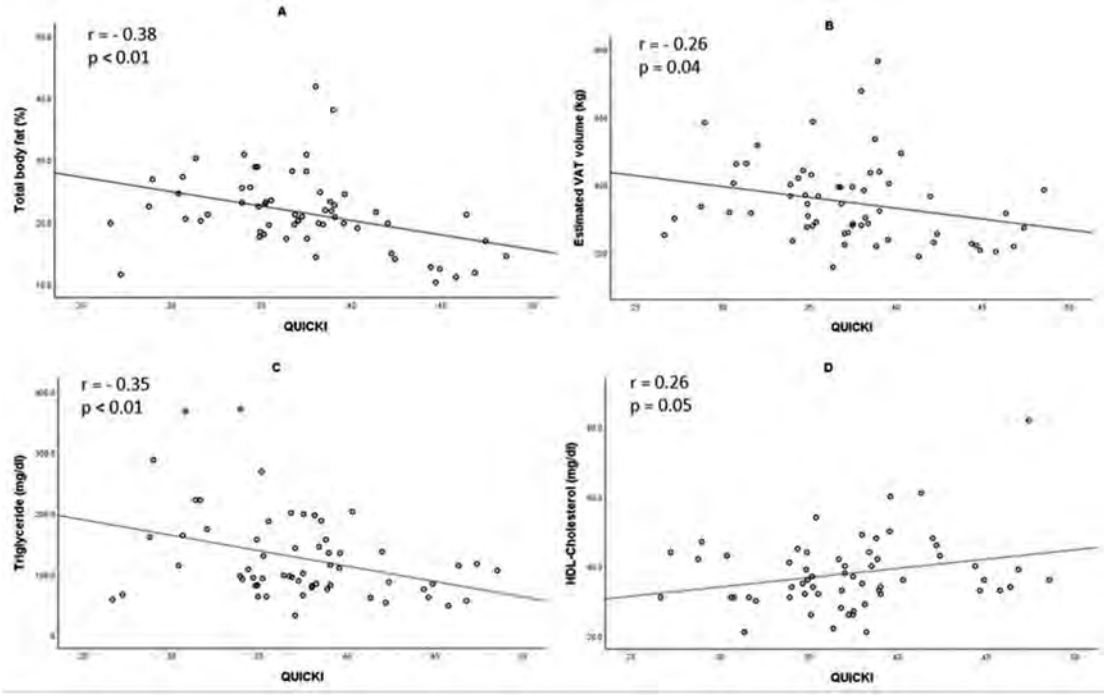

Figure 1 Scatter plots depicting correlation graphs between QUICKI and total body fat (A), estimated VAT volume (kg) (B), Triglyceride (C) and HDL Cholesterol (D).

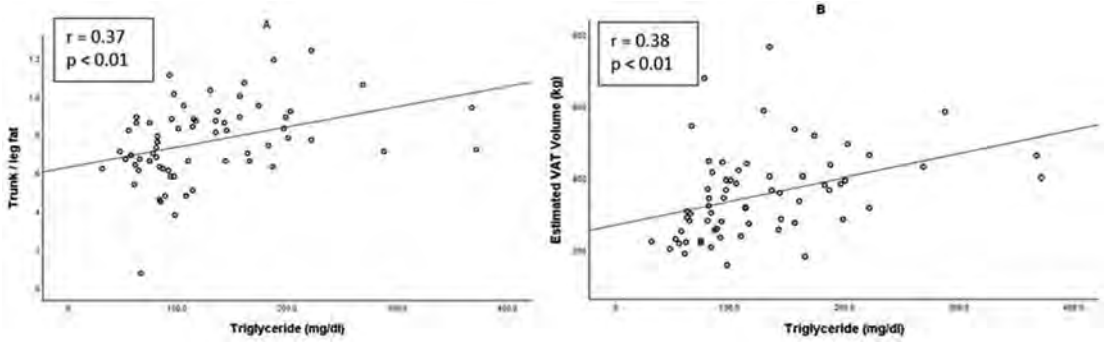

Figure 2 Scatter plots depicting correlation graphs between Triglyceride levels and %trunk/%leg fat ratio (A) and estimated VAT volume (kg) (B).

TG remained significant even after controlling for % total body fat mass ($r = 0.40$, $P < 0.01$). Estimated VAT volume also correlated positively with TG ($r = 0.38$; $P < 0.01$) (Figure 2).

Discussion

This is the first Indian study to comprehensively examine body composition and metabolic profile in the cohort of men with paraplegia. Our study demonstrated that in spite of similar BMIs, subjects with paraplegia had lower lean body mass indices, higher fat mass indices and a poorer metabolic profile when compared to able-bodied individuals.

The definition and cutoffs of obesity and sarcopenia in SCI are poorly defined across literature. The recent review by Silveira *et al.*[31] concludes that neither the BMI, nor waist circumference nor the current body composition cutoffs are validated in SCI population and all the existing cutoffs are defined for able-bodied individuals. For this reason, we have not attempted to classify subjects with SCI as obese or non-obese using a body composition measure cut off, rather presented a continuous measure in comparison with able-bodied individuals.

Our subjects with paraplegia had a lower waist circumference when compared to BMI matched able-bodied controls but higher adiposity, lower lean mass parameters on body composition and higher insulin resistance. This suggests that both BMI and waist circumference are poor markers of metabolic risk in SCI population.

Our study demonstrated lower total lean mass and appendicular lean mass indices in paraplegia group as did many other studies in literature.[6,10,32] In our study, comparison between the MAS categories in subjects with paraplegia showed that the subgroup with higher spasticity had higher lean mass suggesting that spasticity protected against skeletal muscle atrophy. Studies by Gorgey *et al.* also concluded that spasticity defended against skeletal muscle deterioration following SCI.[33,34]

Subjects with paraplegia in our study had a higher fasting blood glucose, higher HOMA-IR and lower QUICKI index as compared to controls. High rates of type 2 diabetes mellitus and fasting hyperglycemia have been previously reported in literature.[24,35] In a previous study done in South India at our center, Tharion *et al.* found that fasting hyperglycemia was present in 19% and glucose intolerance was present in 23% of subjects with SCI.[24] In contrast, studies by Bauman *et al.*[36] and Li *et al.*[37] reported normal fasting glucose but impaired glucose tolerance on an oral glucose tolerance test and have suggested an extrahepatic (likely skeletal muscle) source of insulin resistance.

We found lower HDL-C in our study subjects when compared to able-bodied controls with other lipid parameters not being different. A low HDL-C has been consistently demonstrated in various studies.[16,24,38] While other studies also show an increase in triglyceride and total cholesterol levels,[16,38] both our study and the study by Tharion *et al.*[24] did not find significant difference in other lipid parameters. Meshkini M *et al.* studied ethnic variations in lipid parameters in able-bodied individuals and found that despite a lower BMI and waist circumference, Indians had a lower HDL-C compared to Europeans.[39] HDL-C correlated with truncal adiposity measures and a linear regression model including age, sex, waist circumference, truncal fat percentage and ethnicity explained 53.5% of the variability in HDL-C. On the other hand, dietary intake of fat but not ethnicity was a significant predictor of LDL-C. Genetic and dietary factors were hypothesized to play a role in the ethnic differences in lipid profile.

On correlation analysis, we found that QUICKI correlated with measures of whole-body adiposity but not with lean mass indices. QUICKI also correlated positively with triglyceride levels and negatively with HDL-C levels. This finding is expected as QUICKI is a marker of liver insulin resistance and hence would correlate with adiposity markers and triglyceride levels.[40] The loss of skeletal muscle mass post SCI has been shown to cause decreased peripheral glucose utilization and contributes to lowered peripheral insulin sensitivity [14] which was not measured in our study.

In our study, estimated VAT volume and % trunk/% leg mass correlated positively with triglyceride levels. The latter correlation remained significant even after controlling for whole-body fat mass. Gorgey *et al.* similarly demonstrated that in subjects with SCI regional adiposity contributes directly to the altered metabolic profile.[15]

Our study is only one among a few studies from India [24,25] examining the body composition and metabolic parameters in the population with SCI. We have also tried to demonstrate a correlation between body composition indices and measures of insulin resistance.

Study limitations

Our pilot study has the limitation of having a cross sectional, observational design and small sample size. An oral or intravenous glucose tolerance test would be better able to define the dynamics of glucose handling in this population. Visceral adipose tissue was estimated

from DXA measures though a Magnetic resonance imaging would be ideal. A detailed dietary history would have given greater insight into factors influencing changes in body composition and metabolic outcomes.

Conclusion

Our study in Indian men with chronic paraplegia in comparison to age, and BMI matched able-bodied men demonstrates increased adiposity and decreased lean mass on body composition analysis. The adiposity measures correlated with the worse metabolic profile seen in this group. Body composition rather than BMI is a better way to define adiposity and sarcopenia in this population though well-validated cutoffs are yet to be established.

Disclaimer statements

Contributors None.

Funding None.

Conflict of interest Authors have no conflict of interests to declare

ORCID

Kripa Elizabeth Cherian http://orcid.org/0000-0002-6644-657X

Thomas V. Paul http://orcid.org/0000-0003-3315-341X

References

1 Gater DR. Obesity after spinal cord injury. Phys Med Rehabil Clin N Am 2007 May;18(2):333–51, vii.
2 Jones LM, Legge M, Goulding A. Healthy body mass index values often underestimate body fat in men with spinal cord injury. Arch Phys Med Rehabil 2003 Jul;84(7):1068–71.
3 Laughton GE, Buchholz AC, Martin Ginis KA, Goy RE. SHAPE SCI Research Group. Lowering body mass index cutoffs better identifies obese persons with spinal cord injury. Spinal Cord 2009 Oct;47(10):757–62.
4 Buchholz AC, Bugaresti JM. A review of body mass index and waist circumference as markers of obesity and coronary heart disease risk in persons with chronic spinal cord injury. Spinal Cord 2005 Sep;43(9):513–8.
5 Gorgey AS, Dolbow DR, Dolbow JD, Khalil RK, Castillo C, Gater DR. Effects of spinal cord injury on body composition and metabolic profile – part I. J Spinal Cord Med 2014 Nov;37 (6):693–702.
6 Castro MJ, Apple DF, Hillegass EA, Dudley GA. Influence of complete spinal cord injury on skeletal muscle cross-sectional area within the first 6 months of injury. Eur J Appl Physiol 1999 Sep;80(4):373–8.
7 Wilmet E, Ismail AA, Heilporn A, Welraeds D, Bergmann P. Longitudinal study of the bone mineral content and of soft tissue composition after spinal cord section. Paraplegia 1995 Nov;33(11):674–7.
8 Spungen AM, Wang J, Pierson RN, Bauman WA. Soft tissue body composition differences in monozygotic twins discordant for spinal cord injury. J Appl Physiol Bethesda Md 1985;88(4): 1310–5. 2000 Apr
9 Monroe MB, Tataranni PA, Pratley R, Manore MM, Skinner JS, Ravussin E. Lower daily energy expenditure as measured by a respiratory chamber in subjects with spinal cord injury compared with control subjects. Am J Clin Nutr 1998 Dec;68(6):1223–7.
10 Spungen AM, Adkins RH, Stewart CA, Wang J, Pierson RN, Waters RL, *et al.* Factors influencing body composition in persons with spinal cord injury: a cross-sectional study. J Appl Physiol 2003 Dec;95(6):2398–407.
11 Edwards LA, Bugaresti JM, Buchholz AC. Visceral adipose tissue and the ratio of visceral to subcutaneous adipose tissue are greater in adults with than in those without spinal cord injury, despite matching waist circumferences. Am J Clin Nutr 2008 Mar;87(3):600–7.
12 Bauman WA, Spungen AM. Carbohydrate and lipid metabolism in chronic spinal cord injury. J Spinal Cord Med 2001;24(4): 266–77.
13 Bauman WA, Spungen AM. Metabolic changes in persons after spinal cord injury. Phys Med Rehabil Clin N Am 2000 Feb;11 (1):109–40.
14 Aksnes AK, Hjeltnes N, Wahlström EO, Katz A, Zierath JR, Wallberg-Henriksson H. Intact glucose transport in morphologically altered denervated skeletal muscle from quadriplegic patients. Am J Physiol 1996 Sep;271(3 Pt 1):E593–E600.
15 Gorgey AS, Gater DR. Regional and relative adiposity patterns in relation to carbohydrate and lipid metabolism in men with spinal cord injury. Appl Physiol Nutr Metab 2011 Jan;36(1):107–14.
16 Bauman WA, Spungen AM, Zhong YG, Rothstein JL, Petry C, Gordon SK. Depressed serum high density lipoprotein cholesterol levels in veterans with spinal cord injury. Paraplegia 1992 Oct;30 (10):697–703.
17 Wilt TJ, Carlson KF, Goldish GD, MacDonald R, Niewoehner C, Rutks I, *et al.* Carbohydrate and lipid disorders and relevant considerations in persons with spinal cord injury. Evid Rep Technol Assess (Full Rep) 2008 Jan;163:1–95.
18 Wulan SN, Westerterp KR, Plasqui G. Ethnic differences in body composition and the associated metabolic profile: a comparative study between Asians and Caucasians. Maturitas 2010 Apr;65 (4):315–9.
19 Rush EC, Freitas I, Plank LD. Body size, body composition and fat distribution: comparative analysis of European, Maori, Pacific Island and Asian Indian adults. Br J Nutr 2009 Aug;102(4):632–41.
20 Pan WH, Yeh WT, Weng LC. Epidemiology of metabolic syndrome in Asia. Asia Pac J Clin Nutr 2008;17(Suppl 1):37–42.
21 Deurenberg P, Deurenberg-Yap M. Differences in body-composition assumptions across ethnic groups: practical consequences. Curr Opin Clin Nutr Metab Care 2001 Sep;4(5):377–83.
22 Deurenberg P, Deurenberg-Yap M, Guricci S. Asians are different from Caucasians and from each other in their body mass index/ body fat per cent relationship. Obes Rev 2002 Aug;3(3):141–6.
23 Li J, Polston KFL, Eraslan M, Bickel CS, Windham ST, McLain AB, *et al.* A high-protein diet or combination exercise training to improve metabolic health in individuals with long-standing spinal cord injury: a pilot randomized study. Physiol Rep 2018 Aug;6 (16):e13813.
24 Tharion G, Prasad KR, Gopalan L, Bhattacharji S. Glucose intolerance and dyslipidaemias in persons with paraplegia and tetraplegia in south India. Spinal Cord 1998 Apr;36(4):228–30.
25 Singh R, Rohilla RK, Saini G, Kaur K. Longitudinal study of body composition in spinal cord injury patients. Indian J Orthop 2014 Mar;48(2):168–77.
26 Kirshblum SC, Burns SP, Biering-Sorensen F, Donovan W, Graves DE, Jha A, *et al.* International standards for neurological classification of spinal cord injury (revised 2011). J Spinal Cord Med 2011 Nov;34(6):535–46.
27 Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth Scale of Muscle Spasticity. Phys Ther 1987 Feb;67(2): 206–7.
28 Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985 Jul;28(7):412–9.
29 Katz A, Nambi SS, Mather K, Baron AD, Follmann DA, Sullivan G, *et al.* Quantitative insulin sensitivity check index: a simple, accurate method for assessing insulin sensitivity in humans. J Clin Endocrinol Metab 2000 Jul;85(7):2402–10.
30 Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma,

without use of the preparative ultracentrifuge. Clin Chem 1972 Jun;18(6):499–502.
31 Silveira SL, Ledoux TA, Robinson-Whelen S, Stough R, Nosek MA. Methods for classifying obesity in spinal cord injury: a review. Spinal Cord 2017 Sep;55(9):812–7.
32 Modlesky CM, Bickel CS, Slade JM, Meyer RA, Cureton KJ, Dudley GA. Assessment of skeletal muscle mass in men with spinal cord injury using dual-energy X-ray absorptiometry and magnetic resonance imaging. J Appl Physiol 2004 Feb;96(2): 561–5.
33 Gorgey AS, Dudley GA. Spasticity may defend skeletal muscle size and composition after incomplete spinal cord injury. Spinal Cord 2008 Feb;46(2):96–102.
34 Gorgey AS, Chiodo AE, Zemper ED, Hornyak JE, Rodriguez GM, Gater DR. Relationship of spasticity to soft tissue body composition and the metabolic profile in persons with chronic motor complete spinal cord injury. J Spinal Cord Med 2010;33 (1):6–15.
35 Duckworth WC, Jallepalli P, Solomon SS. Glucose intolerance in spinal cord injury. Arch Phys Med Rehabil 1983 Mar;64(3): 107–10.
36 Bauman WA, Spungen AM. Disorders of carbohydrate and lipid metabolism in veterans with paraplegia or quadriplegia: a model of premature aging. Metabolism 1994 Jun;43(6):749–56.
37 Li J, Hunter GR, Chen Y, McLain A, Smith DL, Yarar-Fisher C. Differences in glucose metabolism among women with spinal cord injury may not be fully explained by variations in body composition. Arch Phys Med Rehabil 2019 Jun;100(6): 1061–7.e1.
38 Nash MS, Mendez AJ. A guideline-driven assessment of need for cardiovascular disease risk intervention in persons with chronic paraplegia. Arch Phys Med Rehabil 2007 Jun;88(6): 751–7.
39 Meshkini M, Alaei-Shahmiri F, Mamotte C, Earnest J. Ethnic variation in lipid profile and its associations with body composition and diet: differences between Iranians, Indians and Caucasians Living in Australia. J Immigr Minor Health 2017 Feb;19(1):67–73.
40 Rankin KC, O'Brien LC, Segal L, Khan MR, Gorgey AS. Liver adiposity and metabolic profile in individuals with chronic spinal cord injury. Biomed Res Int 2017;2017:1364818. doi:10.1155/2017/1364818