ORIGINAL REPORT

SPASTICITY AND BONE DENSITY AFTER A SPINAL CORD INJURY

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Study design: Descriptive, cross-sectional study.

Objective: To assess the relationship between spasticity and bone mineral density in the lower extremities in individuals with a motor complete spinal cord injury.

Methods: Eighteen individuals, matched for time since injury, gender, and age, were included in the study. Nine men had severe spasticity, and 9 men had spasticity that was either mild or not present. Comparisons regarding bone mineral density were made using dual energy X-ray absorptiometry. Regions of interest measured were total leg, pelvis, femoral neck and total hip. Between-group differences regarding fat and lean tissue were analysed.

Results: Background data, such as weight, height, standing and exercising habits, smoking and alcohol use, were similar in both groups. There was no difference between the groups regarding bone mineral density. All of the participants presented with osteoporosis or osteopaenia values at the hips. Participants with severe spasticity had larger muscle volume than those with none or mild spasticity. No correlations between bone mineral density and body composition with age or time since injury were seen.

Conclusion: No difference in bone mineral density dependant on spasticity was detected in this study, but all included participants showed osteopaenia or osteoporosis at the hip, but not in full body values. Individuals with severe spasticity had greater muscle mass compared with those with no or mild spasticity.

Key words: body composition, bone mineral density, DXA, osteoporosis, spasticity, spinal cord injury.

INTRODUCTION

Increased risk of osteoporosis, i.e. fragile bones, is a well-documented problem for wheelchair-dependent individuals with a spinal cord injury (SCI) (1–6). Low-impact fractures have been shown to be more common in the SCI population compared with the able-bodied population (7, 8) where osteoporosis is primarily a problem for women over 50 years of age. The most common explanation for the bone loss is due to immobilization or disuse and to increased bone resorption post-injury. The level of non-loading of the skeleton is related to the degree of demineralization, and therefore it is mainly below the neurological level of injury that demineralization will occur (1, 2, 4, 6). There are multiple causes that can affect bone density; age, gender, menopause, heredity, ethnicity, body height and weight, physical activity, corticosteroid use, calcium intake, smoking, and alcohol (9, 10).

Some authors have discussed whether bone loss in wheelchair-users can be prevented through activities such as weight-bearing through passive standing in a standing frame/tilt board, passive bicycling, or other activities. Two studies (11, 12) showed that early interventions with passive weight-bearing could decrease bone loss early after an SCI, but the long-term results were uncertain. Two other studies showed that passive weight-bearing in standing frames did not have an effect of clinical value on bone density at any sites (13, 14) and the same result has been shown with long leg braces (1). Intensive exercise might prevent bone loss in the upper limbs, but not in the lower extremities (6).

There have been some indications that spasticity might decrease the risk of osteoporosis; however, the results are inconclusive. Spasticity is a complex condition caused by a lesion within the central nervous system. According to previous research, approximately 40% of all individuals with a SCI report problematic spasticity (15). Two studies (16, 17) showed less decrease in bone mineral density (BMD) for persons with a spastic paralysis compared with persons with a flaccid paralysis. On the other hand, 2 further studies (1, 3) reported the opposite, i.e. no difference in BMD between individuals with flaccid or spastic paralysis. Due to the inconsistent results of these studies concerning the influence of spasticity on bone density, the need of well-matched groups and focus on the spasticity factor was needed.

Our research hypothesis was that individuals with strong spasticity have less bone loss than those with a flaccid paralysis in subjects with a motor complete SCI.

The aim of this study was to assess the relationship between spasticity and BMD in the lower extremities in wheelchair-dependent individuals with a motor complete SCI.

MATERIAL AND METHODS

Participants

Eighteen wheelchair-dependent individuals with SCI were included in the study.

Inclusion criteria. Age between 18 and 55 years, diagnosed with a motor complete SCI (American Spinal Injury Association (ASIA) impairment scale (AIS) A or B) (18) for at least 2 years, and ≥16 years of age at the time of injury. Participants should have either strong spasticity; here defined as at least 2 of the included muscle groups graded with a Modified Ashworth Scale (MAS) (19) grade of ≥4, or mild spasticity; here maximum one muscle group with MAS=2 and, the rest MAS=0–1.

Exclusion criteria. Previous or on-going treatment for osteoporosis, medications that can affect bone density, contractures that can challenge bone density assessment, post-menopause, metabolic disease that can cause secondary osteoporosis, present heterotopic ossificans, and pregnancy.

This study was approved by the Regional Ethical Review board (2007/866-31) and the committee of radiation protection at Karolinska University Hospital (34/2007), Stockholm.

Material and methods

This study was undertaken as a descriptive, cross-sectional study. A database search was performed to identify all individuals between the ages of 18 and 55 years with a motor complete SCI. Of those found, all with moderate spasticity (i.e. between the defined criteria for mild and severe spasticity described above) in their last yearly check-up and/or with documented limitations in range of motion (ROM) were excluded. Individuals with either severe spasticity, or no/mild spasticity, were matched for time since injury (±3 years), gender, and age (±7 years). No women were included due to difficulties with appropriate matching. Two participants had deformations in one hip, and those results were excluded from the analysis (Fig. 1).

Spasticity in the hip flexors/extensors/adductors and knee flexors/extensors was assessed by one experienced physical therapist using the MAS (19).

Background data were collected in structured interviews with considerations including; weight-bearing, exercising habits, calcium intake, alcohol and tobacco use, previous or past treatment for spasticity, and history of fractures.

Bone density assessment was performed at the department of radiology with dual energy X-ray absorptiometry (DXA) (Lunar Prodigy Advance, GE Lunar, Madison, Massachusetts, USA). Scans made were total body and bilateral hips in the supine position. The area around the knee was not included in the measurement, due to that site was not available with the machine used. Only data from the lower extremities were analysed due to the variation in weight-bearing of the upper extremities. The lower back was also excluded in the analysis since many of the participants had osteosynthetic material following surgery. BMD was presented in grams/square centimetre (g/cm²) and as a percentage with relation to a reference group that was matched for gender, age and weight.

The World Health Organization (WHO) defined and graded bone mass in 4 steps in 1994 from the DXA examination (20) as:

- *Normal:* a value of BMD within 1 standard deviation (SD) of the young adult reference mean (T-score ≥−1).
- *Osteopaenia (low bone mass):* a value of BMD more than 1 SD below the young adult mean, but less than and 2.5 SD below this value (T-score <−1 and >−2.5).
- *Osteoporosis:* a value of BMD 2.5 SD or more below the young adult mean (T-score <−2.5).
- *Established osteoporosis:* osteoporosis as defined above and one or more fragility fractures.

When DXA-scans are made, information regarding fat and lean mass is included in addition to the bone mineral density result, which is why these data were included in the analysis.

Statistics

SPSS statistical program (Statistical Package for Social Science, version 15.0, Chicago, Illinois, USA) was used in all the analyses. Independent *t*-test was used for all BMD comparisons. The primary outcome measure was the total lower extremities BMD, with results presented as the average value of the right and left sides. Comparisons were also calculated separately for the pelvis, total hip, femoral neck, and total body. Fat and lean tissue, were compared between groups using the independent *t*-test. Participant characteristics and background data were compared with an independent *t*-test, except for level of injury where Fisher's exact test was used due to the small sample size comprising each group. Correlations between time since injury, age, BMD and body composition were analysed using Pearson correlation coefficients. Data are presented as mean (SD), and the level of significance was accepted at $p < 0.05$.

RESULTS

There were no differences in the participant characteristics between the groups (Table I).

Background data were similar between groups. Two persons in each group performed regular standing training. Level of physical activity varied in both groups from no training to competition level training. One person in each group was a smoker. Alcohol was occasionally to moderately used by 6 individuals in each group. Calcium intake was according to each
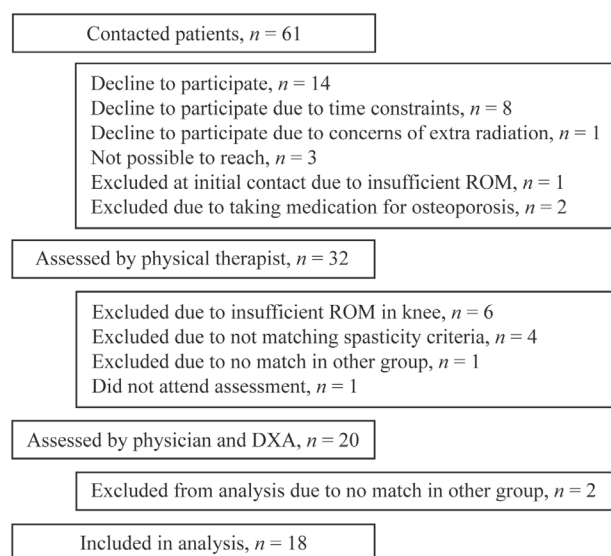

Fig. 1. Inclusion/exclusion process. ROM: range of motion; DXA: dual energy X-ray absorptiometry.

Contacted patients, *n* = 61

Decline to participate, *n* = 14
Decline to participate due to time constraints, *n* = 8
Decline to participate due to concerns of extra radiation, *n* = 1
Not possible to reach, *n* = 3
Excluded at initial contact due to insufficient ROM, *n* = 1
Excluded due to taking medication for osteoporosis, *n* = 2

Assessed by physical therapist, *n* = 32

Excluded due to insufficient ROM in knee, *n* = 6
Excluded due to not matching spasticity criteria, *n* = 4
Excluded due to no match in other group, *n* = 1
Did not attend assessment, *n* = 1

Assessed by physician and DXA, *n* = 20

Excluded from analysis due to no match in other group, *n* = 2

Included in analysis, *n* = 18

Table I. *Participant characteristics, presented as mean (SD) [range], for individuals with severe spasticity (n = 9) and individuals with none or mild spasticity (n = 9)*

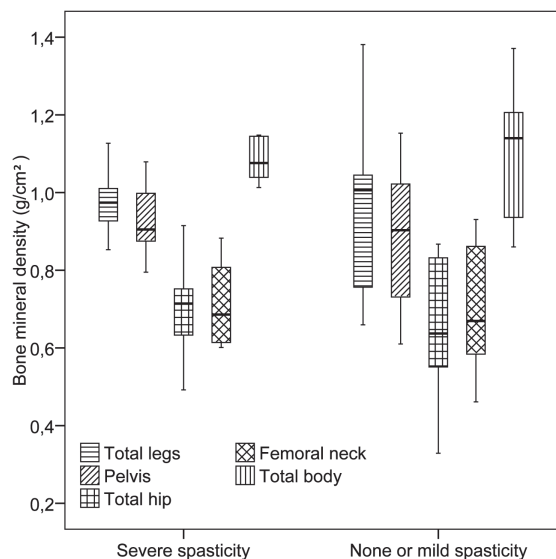| Participant characteristics	Severe spasticity	None/mild spasticity	*p*-value
Age, years	37 (7.9) [24–49]	39 (8.9) [28–53]	0.602
Age at injury, years	22 (3.6) [16–27]	25 (4.1) [19–32]	0.130
Time since injury	15.2 (8.1) [5–27]	14.4 (8.5) [4–27]	0.845
Height, cm	184 (5.4)	182 (6.9)	0.682
Weight, kg	77.8 (18.0)	78.7 (14.3)	0.909
BMI	23.0 (5.0)	23.6 (3.7)	0.787
Para/tetraplegia, *n*	6/3	8/1	0.570

BMI: body mass index; SD: standard deviation.

Fig 2. Comparison between groups of bone mineral density (BMD) for total leg, pelvis, total hip, femoral neck, and total body. Data are presented as median values with inter-quartile ranges.

individual's estimation sufficient in 7 participants with severe spasticity and 5 in those with none/mild. Fractures after the onset of SCI had occurred in one out of 9 persons in the severe spastic group and 2 out of 9 persons in the no or mild spasticity group, and none reported low impact fractures. One person in each group used medication for spasticity management.

There was no difference between the groups regarding BMD measurements (Fig. 2). No subgroup analysis regarding persons with tetra/paraplegia was performed due to too few individuals with tetraplegia ($n=4$). Little, if any, correlation ($r \le 0.25$) (Monro's descriptive terms for the strength of correlation) (21) between BMD and age or time since injuries were found.

Osteoporosis values at the hips were present in 67% of participants and the rest showed osteopaenic values. The results were similar in the femoral neck, but not for full-body values where osteoporosis was present in 28% of participants. Little, if any, correlation (21) between osteoporosis and time since injury was found ($r=0.242$).

The results showed a difference in muscle mass, with participants with severe spasticity having larger muscle mass than those with none or mild spasticity, $p=0.004$, while no difference was seen in fat tissue, $p=0.542$ (Table II). Little, if any, correlation ($r \le 0.25$) (21) was found between lean and fat tissue, and between lean and bone tissue for total hip. Correlation between lean or fat tissue with age or time since injury was found to be non-significant.

DISCUSSION

A few studies have investigated the relationship between BMD and spasticity. Our finding, that spasticity does not preserve bone mass in individuals with a motor complete SCI, is supported by 2 studies (1, 3). Biering-Sörensen et al. (1) compared 6 individuals with spastic paraplegia with 10 individuals with a flaccid paresis and found no difference in BMD. In their study, participants had an AIS B or C, i.e. some participants had voluntary motor function below the level of injury. However, all included participants were wheelchair-users. The other study (3) included 31 individuals with complete and incomplete paraplegia in a prospective design where 15 participants developed spasticity during the study period (5–50 weeks post-injury). No difference between individuals with a flaccid or spastic paralysis was found. On the contrary, 2 studies suggest that spastic paralysis may indeed preserve bone mass (16, 17). In one of these (16), where approximately 50% of the study cohort had an incomplete injury, bone loss was more prevalent in those with a flaccid paralysis compared with those with a spastic paralysis. The other study (17), including 54 participants with a spastic and 6 participants with a flaccid SCI, showed that spasticity had a preserving effect on bone density in the femoral shaft and femoral distal epiphysis, but not in the lower leg. The strength of our study was that all included participants had a motor complete injury, were matched for possible confounding factors, and there was a large difference in grade of spasticity between the 2 groups.

The lower extremities are more affected regarding bone loss than the upper extremities, especially for persons with paraplegia who have normal or increased BMD in the upper extremities due to the increased load (1, 2, 4, 16). Demirel et al. (16) reported that there was no difference regarding bone density in the lower extremities between individuals with paraplegia and tetraplegia. We decided to analyse only the lower extremities, since the level of activity and weight bearing differ to a large extent in the upper extremities in SCI wheelchair-users due to differences in level of injury. The results of our study showed no between-group difference for BMD or osteoporosis values, but we did find osteopaenia or osteoporosis values in the hip-region in all of the participants.

Several studies have shown that the greatest bone loss occurs during the first 2 years after injury and reaches a steady state at a lower level than in the normal population (2, 22). However inconclusive, we chose to include only participants who had been injured for 2 years or more and, additionally, time since injury was made the most important match-factor.

Decreased lean body mass is a well-known problem within the SCI-population and may lead to an increased risk of metabolic abnormalities, such as carbohydrate intolerance, insulin

Table II. *Between-group comparison of body composition, measured in gram, for total lower extremities presented as mean (SD) [range]*

	Severe spasticity $n=9$	None/mild spasticity $n=9$	*p*-value
Lean tissue	14,635 (2,815) [10,388–18,854]	10,911 (1,797) [8,462–13,009]	0.004
Fat tissue	6,919 (3,502) [1,832–11,798]	7,879 (3,022) [1,701–11,259]	0.542

SD: standard deviation.

resistance, lipid abnormalities, and heart disease. These are common in the able-bodied population, as age-associated disorders, but occur prematurely and at a higher prevalence in the population with SCI (23, 24). Our study showed a difference in muscle mass with the spastic group having larger lean tissue volume, which has also been shown by a recent study (25). This is an important finding, since the greater muscle mass might moderate the risk of metabolic disease that comes with living with a SCI.

We did not detect any correlation between lean and bone tissue. These data are supported by Bauman et al. (4), who showed that lean tissue influenced bone density in the able-bodied population, but not in the SCI-population. This might be due to the fact that it is not only the muscle mass itself that is important for maintaining BMD, but also to what degree there is voluntary muscle function. This hypothesis was also raised by Biering-Sörensen et al. (2), who found less decrease in hip-BMD in one participant with an L1 injury, where muscle function is preserved, than in participants with higher levels of injury. The same finding was seen in our study, where the highest BMD measurement was found in the participant with the lowest neurological level (L1).

In our study, we did not detect any correlation between fat and lean tissue, nor a difference in fat mass between groups. Even if not shown in our study, several previous studies have shown that increased fat mass or obesity is more common in the SCI population compared with the able-bodied population (23, 26, 27). Obesity might lead to a lower functional outcome, more difficulties with transfers and need of more assistance, as well as increased risk of medical conditions such as carpal tunnel syndrome, pulmonary embolism and obstructive sleep apnoea (27).

Absence of spasticity may lead to increased risk of weight gain, as energy expenditure will decrease with less spasticity. While not shown here, a reduction in energy requirements to maintain weight was observed following intrathecal administration of the anti-spasticity agent Baclofen in a case study of a boy with mental retardation, fed through gastrostomy (28).

It has been shown that the main BMD loss after an SCI occurs in the proximal tibia (50% of normal BMD value) and the femoral neck (25% of normal BMD value) (1, 2). The proximal tibia is a common site of measurement, in addition to the regions used in this study, when making DXA scans on participants with an SCI. One limitation in our study was that this site was not available at the time of the study with the DXA machine used. Another limitation in our study is lack of power. However, both groups were well matched regarding age and time since injury, and all participants had motor complete injuries. Participant groups were alike with regard to demographic characteristics and background data. Weight has been shown to have a great impact on bone density and might have been more appropriate to have as a matching criterion than gender. There were no women included in our study, due to difficulties in proper matching.

Two persons were taking anti-spasticity medication, one in each group. Several participants with severe spasticity in our study expressed benefits from their spasticity and had stopped taking anti-spastic medication some years after injury. It has been shown that the subjective negative aspect of spasticity decreases with time since injury (29), which might explain the low number of persons medicating for their severe spasticity.

Our research hypothesis, that spasticity can influence bone mass, was not confirmed in this study. Further research regarding prevention of bone loss due to immobilization is still needed in order to create guidelines for assessment and management of osteoporosis within the SCI population. This is of great interest considering the aging SCI population group and is therefore a growing area of concern.

In conclusion, no difference in BMD depending on level of spasticity was found in individuals with a motor complete SCI; however, osteoporosis/osteopaenia at the hip, but not in full body values was observed in all participants. Individuals with severe spasticity had greater muscle mass compared with those with no or mild spasticity.

ACKNOWLEDGEMENTS

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