

## Spasticity and bone mineral density after spinal cord injury

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### Abstract

**Objective:** To evaluate the effects of lower limb spasticity on bone mineral density (BMD) after chronic spinal cord injury. **Design:** Observational cross-sectional study. **Setting:** Department of PMR, SMS hospital, Jaipur. **Participants:** 50 individuals of chronic motor complete SCI were classified into mild (n=16), moderate (n=11), and severe (n=23) spastic groups; based on their lower limb extensor muscle group spasticity score using a Modified Ashworth Scale (M.A.S). A DEXA scanner was used to measure bone mineral density (BMD, g/cm<sup>2</sup>) and were compared between the groups with different grades of spasticity. **Results:** The mean M.A.S score in severe, mild and moderate spastic was 5.28 ± 0.54, 1.84±0.30 and 3.14±0.32 respectively (P<0.001S). Majority i.e. 46% (n=23) were osteopenic ; 16% (n=8) were osteoporotic; while 38% (n=19) had normal bone mineral density at hip joint (P=0.753NS). The mean duration of spinal cord injury was 2.14±0.881 years (P = 0.487NS). **Conclusion:** A significant association was found between mean M.A.S and type of spasticity. While no significant association was observed between the severity of lower limb spasticity and bone mineral density among SCI individuals.

**Keywords:** SCI: Spinal cord injury, M.A.S: Modified Ashworth scale.

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### Introduction

Spinal cord injury (SCI) may lead to a partial or complete loss of neural transmission below the level of spinal cord injury. Internationally, road traffic accidents are the overall most common cause of SCI. High falls associated spinal cord injuries are more common in the younger population whereas low falls associated spinal injuries are more common in the older population due to associated osteoporosis[1,2]. The complications associated with spinal cord injury include respiratory complications, spasticity, pressure ulcers, deep vein thrombosis, neurogenic bladder and bowel, heterotopic ossification, osteoporosis, muscle wasting, endocrinologic and metabolic changes such as adiposity, a state of insulin resistance,

hyper-insulinemia, hyperlipidemia, and hypertension [3,4]. Spasticity defined by Lance as, "It is a motor disorder characterized by a velocity dependant increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper excitability of the stretch reflex, as one of the component of the upper motor neuron syndrome"[5]. Symptoms of spasticity are experienced by the 65-78% of individuals with chronic (≥1-year post-injury) SCI and are a possible contributor to reduced quality of life (QOL)[4,6]. Osteoporosis which leads to fragile bones and predispose individuals to bone fractures, is a well-known chronic complication of non ambulatory individuals with spinal cord injury (SCI). The most common explanation for the loss in bone mass in chronic spinal cord injury patients is due to increased bone resorption and immobilization or disuse after the spinal cord injury. Bone loss is mainly below the neurological level of injury because demineralization is related to the level of non loading of the skeleton[7]. Spasticity might decrease the risk of osteoporosis; however literature depicts variable effects of spasticity on bone mineral density and the results are

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still inconclusive as few studies showed less decrease in BMD in spastic individuals as compared to those with flaccid paralysis, while some studies have shown no difference in BMD among the two groups. Eser P et al (2005) concluded that spasticity had a preserving effect on bone density in the femoral shaft and femoral distal epiphysis, but not in the lower leg[8], while Jung IY et al (2017) observed no difference in bone mineral density in severely spastic individuals as compared to mild or no spastic group.[9].The objective of the present study amid an emerging interest of general clinicians and rehabilitation specialists over worldwide to combat the associated metabolic health consequences after spinal cord injury, was to evaluate the effects of lower limb spasticity upon bone mineral density (BMD) in chronic SCI

## Material and methods

### Study design, setting, and participants

This was a observational cross-sectional study conducted on spinal cord injured individuals having spasticity in the lower limbs admitted in the Department of Physical Medicine and Rehabilitation of SMS hospital, Jaipur between May 2018 to April 2019. The study was approved by the institute human ethics committee. A total of 50 chronic (duration  $\geq 1$  year), motor complete (ASIA scale A or B), spinal cord injured individuals having spasticity in lower limbs, aged between 18-60 years; BMI between 15- 30 kg/m<sup>2</sup> and those who gave informed written consent were **included** in the study. Patients with a previous history of any co-morbid medical or surgical condition , having treatment history with drugs affecting bone

metabolism such as long term glucocorticoids , cancer drugs, thyroid hormone etc, interventional treatment for spasticity, chronic smokers or alcoholics, and other clinical conditions associated with spasticity such as cerebral palsy, traumatic brain injury, stroke, etc were excluded from the study.

### Evaluation of lower limb spasticity (as per M.A.S)[10].

A detailed history, clinical examination, and relevant investigations of recruited cases were performed in the initial workup. Clinical assessment and determination of the neurological level were done according to the ASIA impairment scale. The Modified Ashworth Scale (M.A.S)[10] for the knee extensors and ankle extensors was used to evaluate lower extremity spasticity in the supine position. It was measured at the same time of the day (between 8 AM to 9 AM) for all cases. To evaluate spasticity, MAS 1+ was converted to grade 2 and subsequently, MAS grades 2, 3, 4 were changed to 3, 4, and 5 respectively. The score of EMAS extensor muscle group was calculated using Equations: Eq. (1) to (3), as done in a study by Jung IY et al[9].

**Eq.:1.** Avg. knee extensor (MAS score):

$$\frac{\text{Right knee extensor} + \text{left knee extensor MAS score}}{2}$$

**Eq.:2.** Avg. ankle extensor (MAS score):

$$\frac{\text{Right ankle extensor} + \text{left ankle extensor MAS score}}{2}$$

**Eq.:3.** Total MAS ( $\Sigma$ MAS) score: - Avg. knee ext. (MAS) score + Avg. ankle ext. (MAS) Score  
 $\Sigma$ MAS extensor muscle group score ranges from 0 to 10; study subjects were classified into mild ( $\Sigma$ MAS score of  $\leq 2$ ), moderate ( $\Sigma$ MAS score of  $> 2$  and  $< 4$ ) and severe ( $\Sigma$ MAS score  $\geq 4$ ) spastic groups.

**Table 1: Six grades of the modified Ashworth scale**

|         |   |   |
|---------|---|---|
| Grade 0 | - | No increase in muscle tone.   |
| Grade 1 | - | Slight increase in muscle tone, manifested by a catch or by minimal resistance at the end of the ROM, when the affected part(s) is moved in flexion or extension. |
| Grade 2 | - | Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM.                       |
| Grade 3 | - | More marked increase in muscle tone through most of the ROM, but the affected part(s) can be easily moved.  |
| Grade 4 | - | Considerable increase in muscle tone, and passive movement is difficult.  |
| Grade 5 | - | Affected part(s) is rigid in flexion or extension.  |

## Evaluation of bone mineral density (BMD)

A Dual-Energy X-ray Absorptiometry Scanner (HOLOGIC – Explorer QDR series) was used to measure bone mineral density (BMD,  $\text{g}/\text{cm}^2$ ). The Subjects were classified for grade of bone mineral density at hip joint in 4 steps using the World Health Organization (WHO) definition[11].

1. Normal: a value of BMD within 1 standard deviation (SD) of the young adult reference mean (T score  $\geq -1$ ).
2. Osteopenia (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  $> -2.5$ ).
3. Osteoporosis: a value of BMD 2.5 SD or more below the young adult mean (T score  $< -2.5$ )
4. Established osteoporosis: osteoporosis as defined above and one or more fragility fractures.

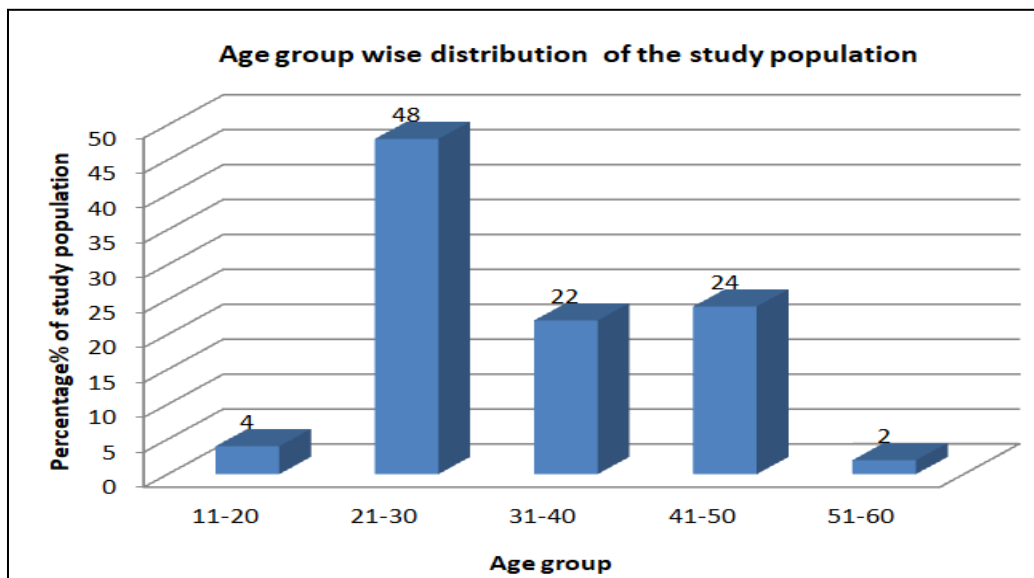
**Outcomes Variables:** The various proportion of cases in mild, moderate, severe spastic groups, the mean M.A.S score using Modified Ashworth Scale and the mean BMD using DEXA (Dual Energy X-ray Absorptiometry) scanner machine (Hologic-Explorer).

**Statistical Analysis:** Taking into account the age, gender, diet, addiction, socioeconomic status, environmental status and ensuring that all three groups

were undergoing similar exercise regimes, the three group data were compared statistically. Statistical Package for Social Sciences (SPSS trial version 23.0) was used for statistical analysis. The qualitative data were expressed in proportion and percentages and the quantitative data expressed as mean and standard deviations. The difference in proportion was analyzed by using the chi-square test. The difference in means among the groups was analyzed using the ANOVA (Analysis of variance test). Correlation between quantitative outcomes was assessed using the Pearson correlation coefficient. The significance level for tests was determined as 95% ( $P < 0.05$ ).

## Results

Within the study group, 46% ( $n=23$ ) had severe spasticity, 32% ( $n=16$ ) were mild spastic while 22% ( $n=11$ ) had moderate spasticity in lower limbs. Majority, 78% ( $n=39$ ) were neurologically complete (ASIA A), 70% cases ( $n=35$ ) belonged to the young age group between 21- 40 years, 84% ( $n=42$ ) were males and 72% ( $n=36$ ) were married.



**Fig 1: Age group distribution of the study population**

The mean M.A.S score among the severe spastic group was  $5.28 \pm 0.54$ . The mean M.A.S score among the mild spastic group was  $1.84 \pm 0.30$  while it was  $3.14 \pm 0.32$  among the moderate spastic group. The

overall mean M.A.S score among the study group was  $3.71 \pm 1.60$ . A significant association was found between  $\sum$  MAS EXT and type of spasticity ( $P \leq 0.001$ ) in the study group.

**Table 2: Association of  $\Sigma$  MAS EXT score with the different grade of spasticity**

|                        |              | N         | Mean        | Std. Deviation | ANOVA   | 1 vs 2  | 1 vs 3  | 2 vs 3  |
|------------------------|--------------|-----------|-------------|----------------|---------|---------|---------|---------|
| $\Sigma$ MAS EXT score | Mild         | 16        | 1.84        | 0.30           | <0.001S | <0.001S | <0.001S | <0.001S |
|                        | Mod          | 11        | 3.14        | 0.32           |         |         |         |         |
|                        | Severe       | 23        | 5.28        | 0.54           |         |         |         |         |
|                        | <b>Total</b> | <b>50</b> | <b>3.71</b> | <b>1.60</b>    |         |         |         |         |

Maximum number of SCI patients i.e. 46% (n=23) were osteopenic while only 16% (n=8) were osteoporotic and 38%(n=19) had normal bone mineral density at hip joint . It was observed that only 13.04% of severe spasticity group had osteoporosis while 18.18% of moderate and 18.75% of mild spasticity

group had osteoporosis. However, this result was statistically non significant(P= 0.753). The mean duration of spinal cord injury in our studied population was 2.14±0.881 years. No significant association between mean duration of spinal cord injury with bone density was found (P = 0.487NS).

**Table 3: Association of Bone mineral density with the grade of spasticity**

| Bone Mineral Density | MILD |       | MOD |        | SEV |        | Grand Total |
|----------------------|------|-------|-----|--------|-----|--------|-------------|
|                      | No   | %     | No  | %      | No  | %      |             |
| Normal               | 7    | 43.75 | 5   | 45.45  | 7   | 30.43  | 19          |
| Osteopenia           | 6    | 37.5  | 4   | 36.36  | 13  | 56.52  | 23          |
| Osteoporosis         | 3    | 18.75 | 2   | 18.18  | 3   | 13.04  | 8           |
|                      | 16   | 100   | 11  | 100.00 | 23  | 100.00 | 50          |

(Chi-square = 1.907 with 4 degrees of freedom; P = 0.753)

**Table 4: Distribution of the cases according to mean duration of injury**

| Bone density | No. of cases | Mean duration of injury | Std. Deviation | P value LS |
|--------------|--------------|-------------------------|----------------|------------|
| Normal       | 19           | 2.00                    | .882           | 0.487NS    |
| Osteopenia   | 23           | 2.30                    | .876           |            |
| Osteoporosis | 8            | 2.00                    | .926           |            |
| <b>Total</b> | <b>50</b>    | <b>2.14</b>             | <b>.881</b>    |            |

### Discussion

The current study observed the influence of spasticity in chronic spinal cord injury patients on the bone mineral density at hip joint. Although the life expectancy of persons with spinal cord injury has optimized firmly as a preventive and treatment approach has enriched over the past decades, evolution of spasticity in such individuals can lead to limitations in daily functions due to pains, fatigue and may cause frequent falls inducing decreased self-esteem and deterioration in the perceived quality of life. In spite of its adverse influences ,spasticity also exerts some favorable and beneficial effects on sitting, standing, transfers, assists ADLs, and maintaining body metabolism and favorable body composition. Present study observed among SCI individuals ; 84% (n=42) were males, primarily young earning age group (n=35)

and married(n=36) , which lead to emotional , social and huge financial stress to the whole family, hampering their physical quality of life , particularly in developing countries such as India. Thus, evolves the need of hour, to create awareness and use of protective measures while being on work, to prevent such kind of devastating injuries. Present study observed that maximum number of SCI patients were in osteopenic group; most of them were in severe spastic group and minimum number of osteoporotic patients were present in severe spastic group as compared to mild and moderate spastic group. This suggests that spasticity may be exerting some protective role on the bone mineral density although this result was statistically nonsignificant. (P = 0.753). So we could conclude that there was no significant association observed between the grading of spasticity with bone mineral density

( $P=0.753$ ). The results of prior studies conducted to find out the influence of spasticity on bone density have been variable. Present study result is similar to the study done by Lofvenmark I et al[7] which assessed the relationship between spasticity and bone mineral density in the lower extremities in nine mild or no spastic and nine severe spastic individuals with a motor complete spinal cord injury and observed no difference for bone mineral density among the two groups. They also reported that participants presented with osteoporosis or osteopenia values at the hips and no correlation amidst bone mineral density and body composition with age or time since injury was seen. Similarly Maggioni M et al[12] conducted a body composition assessment study on thirteen sedentary spinal cord injury subjects and 13 able-bodied healthy males and concluded that total BMD did not differ between the SCI and control(C) groups. Frey RP et al[13] also concluded that the intensity of physical activity did not significantly influence the loss of BMD in all subjects with paraplegia and tetraplegia. However, in few subjects, regular intensive loading exercise activity in early rehabilitation (tilt table, standing) can possibly attenuate the decrease of BMD of tibia however no influence was found for the degree of spasticity on the bone loss in all subjects with SCI. Wilmet E et al[14] observed a rapid decline in bone mineral content (BMC) in the paralyzed areas and that the bone mineral content does not return to pre-injury values within 1 year, thence concluded that there should be an interest in preventing bone loss early in the course of the disease. Biering SF et al[15] compared 6 individuals with spastic paraplegia with 10 individuals with a flaccid paresis and found no variation in bone mineral density (BMD). Recently Jung IY et al[9] compared the mild and severe spastic SCI group for bone marrow density (BMD) and found that the BMD did not differ between the two groups. In contrast to our study results, Demirel G et al[16] assessed bone mineral density (BMD) in both the upper and lower extremities following SCI and found a significant difference in BMD between upper and lower extremities of the paraplegics, however, BMD of upper and lower extremities were similar in tetraplegics and the BMD scores of the lower extremities were similar in the two groups. The decrease in BMD was less in the spastic patients when compared to the flaccid group. A positive correlation was found between time from injury and the degree of BMD deficit in the paralyzed areas, however in present study flaccid paralysis patients were not included. Eser P et al[8] also supported this fact by mentioning that spasticity had a preserving effect on bone density in the

femoral shaft and femoral distal epiphysis, but not in the lower leg. The mean duration of post spinal cord injury in our study was  $2.14 \pm 0.881$  years and no significant correlation between the BMD and duration since injury was seen. This result was similar to the study done by Lofvenmark I et al[7], where no correlation between bone mineral density and body composition with age or time since injury was seen. To conclude, no significant association was observed between the grading of lower limb spasticity and bone mineral density ( $P=0.753$  NS), or mean post duration of spinal cord injury ( $P = 0.487$ NS) among the spinal cord injury patients. The strength of present study was a relatively large sample size ( $n=50$ ) than previous studies, all included participants had a motor complete injury, and were on similar exercise regimes. Future large sample group studies are further required for in-depth knowledge on effects of spasticity on BMD in an attempt to prevent bone loss early in the course of spinal injury.

#### Abbreviations

SCI: Spinal cord injury, M.A.S: Modified Ashworth scale.

#### References

1. Wyndaele M, Wyndaele JJ. Incidence, prevalence and epidemiology of spinal cord injury. what learns a worldwide literature survey? *Spinal Cord* 2006; 44(9):523–9.
2. Chiu WT, Lin HC, Lam C, Chu SF, Chiang YH, Tsai SH. Review: epidemiology of traumatic spinal cord injury: comparisons between developed and developing countries. *Asia Pac J Public Health* 2010; 22(1):9–18.
3. Adams MM, Hicks AL. Spasticity after spinal cord injury. *Spinal Cord* 2005; 43(10):577–586.
4. Sköld C, Levi R, Seiger A. Spasticity after traumatic spinal cord injury: nature, severity, and location. *Arch Phys Med Rehabil.* 1999; 80: 1548–1557.
5. Lance, JW. The control of muscle tone, reflexes, and movement: Robert Wartenberg Lecture. *Neurology* 1980; 30: 1303–1313.
6. Maynard FM, Karunas RS, Waring WP. Epidemiology of spasticity following traumatic spinal cord injury. 3rd. *Arch Phys Med Rehabil.* 1990; 71(8):566–569.
7. Lofvenmark I, Werhagen L, and Norrbrink C. Spasticity And Bone Density After A Spinal Cord Injury. *J Rehabil Med* 2009, 41: 1080–1084. : s.n.

8. Eser P, Frotzler A, Zehnder Y, Schiessl H, Denoth J. Assessment of Anthropometric, Systemic, and Lifestyle Factors Influencing Bone Status in the Legs of Spinal Cord Injured Individuals. *Osteoporos Int* 2005;16 (1):26-34,
9. Jung IY, Kim HR, Chun SM, Leigh JH Shin HI. Severe spasticity in lower extremities is associated with reduced adiposity and lower fasting plasma glucose level in persons with spinal cord injury. *Spinal Cord* 2017; 55: 378–382.
10. Bohannon RW, Smith MB. Inter rater reliability of a Modified Ashworth Scale of muscle spasticity. *Phys Ther.*1987;67(2):206-207.
11. Organization, Report of a WHO Study Group. Assessment of fracture risk and its application for screening for post menopausal osteoporosis. Geneva: World Health and 1994 WHO technical Report series, no.843.
12. Maggioni M, Bertoli S, Margonato V, Merati G, Veicsteinas A, Testolin G .Body Composition Assessment in Spinal Cord Injury Subjects.. *Acta Diabetologica* ; 2003 :40 Suppl 1, S183-6. : s.n.
13. Frey Rindova P, De Bruin ED, Stüssi E, Dambacher MA, Dietz V. Bone Mineral Density in Upper and Lower Extremities During 12 Months After Spinal Cord Injury Measured by Peripheral Quantitative Computed Tomography. *SpinalCord.*2000 ;38(1):26-32..
14. 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;33 (11):674-7.
15. FBH, Biering-Sorenson. Bone mineral content of the lumbar spine and the lower extremities years after spinal cord lesion. *Paraplegia* 1988;26:293-301.
16. Demirel G, Yilmaz H, Paker N, Onel S. Osteoporosis After Spinal Cord Injury. *Spinal Cord*, 1998;36(12):822.

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