ISSN: 2470-4539

Gratis

Journal of Bone Biology and Osteoporosis

Editorial

How to Define Bone Loss in Spinal Cord Lesion

Yannis Dionyssiotis*

Department of Orthopaedics, General University Hospital, ATTIKON, Athens, Greece

Received: April 07, 2015; Accepted: April 09, 2015; Published: April 10, 2015

Editorial

Osteoporosis, the most common of all metabolic bone disorders, is defined by the World Health Organization (WHO) as "a skeletal disease, characterized by low bone mass and micro-architectural deterioration of bone tissue, with a consequent increase in bone fragility and susceptibility to fracture" [1-3]. WHO created an operational definition of postmenopausal osteoporosis based on T and Z score measurements of Bone Mineral Density (BMD) with dual energy X-ray absorptiometry (DXA). According to the ranking system of WHO postmenopausal women are categorized as: 1) normal: if BMD value is of not less than one Standard Deviation (SD) than the average young adult's (T-score>-1), 2) osteopenic: if BMD value is between one and 2.5 SD below the average for young adults (-1<T-score<-2.5), 3) osteoporotic: if BMD value is 2.5 SD or more below the average for young adults (T-score>-2.5) and 4) women with severe or established osteoporosis: if BMD value is 2.5 SD or more below the average for young adults and one or more fractures are present [3,4]. A similar categorization exists for Z-scores. The Z-score is the number of standard deviations above or below what is normally expected for someone of similar age, sex, weight and race in question. In clinical practice, it is useful because a Z-score of below -1.5 SD probably indicates secondary causes of osteoporosis [5].

The clinical usefulness of the DXA T-score in diagnosing low bone density in subjects with a lesion of spinal cord remains unclear. Despite the increased number of risk factors in spinal cord injury or lesion which make a disabled person at increased risk of low bone mass, guidelines are not available on BMD measurements and, as well, virtually no ranking system exists similar to the one already mentioned for postmenopausal women. For this reason, it would be more appropriate for young men and premenopausal women with spinal injuries to use the Z-score obtained from the measurement of bone densitometry for the assessment of bone density. But this remains a simple approach in a difficult problem and maybe is not the case in the so called "bone loss in SCI". It is wrong to categorize loss of bone after spinal cord injury or lesion as secondary immobilization osteoporosis because there is no comparison between these two conditions. Bone loss percent after SCI is much higher as the 10% of immobilization osteoporosis and moreover could be never reversed with the current countermeasures of all types (physical modalities and pharmaceutical). However, most authors are continuing to publish papers using terms such osteoporosis, bone loss, and osteopenia and subjects are classified in these publications according to WHO's criteria for postmenopausal women [6].

Back in 1997 Frost et al. already explained why

***Correspondence:** Yannis Dionyssiotis, MD, PhD, FEBPRM, Department of Orthopaedics, General University Hospital, ATTIKON, Athens, Greece, Tel: +306946469759; E-mail: yannis_dionyssiotis@hotmail.com

the traditional definitions of osteoporosis and osteopenia are not appropriate to describe bone loss in all diseases and explained the regional acceleratory phenomenon and mechanostat theory [7]. Even the term "bone loss" is probably not specific enough, and might imply that the change in bone status is being recorded over time. The explanation lies in differences between traumatic and pathological lesions of the spinal cord accompanied by ongoing loss of bone. There are a variety of mechanisms according to the progression or not of the disease (i.e. progressive multiple sclerosis vs. complete paraplegia), the type of injury (i.e. lesion with a level of injury vs. upper motor neuron pyramidal lesion), life expectancy, the residual mobility and functionality, the ability to walk and stand (i.e. incomplete paraplegia vs. quadriplegia vs. high-low paraplegia) and drug treatment (i.e. frequent corticosteroid therapy in multiple sclerosis vs. long-term therapy with anticoagulants in paraplegia). In addition, there are differences in the degree of spasticity (i.e., flaccid vs. spastic paralysis) and it is necessary to take into account the issue of fatigue and muscle weakness in disabilities. Moreover, patients with motor disorders often face problems of depression which can make it difficult to comply with the proposed treatments by the physicians and limit mobility. According to physical disability there are differences between complete (an absence of sensory or motor function below the neurological level, including the lowest sacral segment) and incomplete lesions (partial preservation of motor and/or sensory function below the neurological level, including the lowest sacral segment). Patients with complete injuries have greater bone loss than those with an incomplete injury and as has already been shown in Brown-Sequard subjects (incomplete SCI) where BMD of the more paretic knee was lower than that of the stronger knee [8,9].

However, in spinal cord lesions there are also similarities; for example the clinical equivalence of diseases with different physiopathology, location, evolution, etc.: a severe form of Multiple Sclerosis (MS) can result in a wheelchair bound patient having a clinical figure equivalent to paraplegia; a patient with MS may have better walking gait pattern in comparison with a patient with incomplete paraplegia but may also be unable to walk, bedridden and vice versa [6]. In addition to these differences and similarities the role of factors which do not change, i.e., race or gender of patients is inadequately clarified. Few studies of women with disabilities debate that bones are more affected than that of men with disabilities. There is a tendency for chronic SCI women to have lower bone mass than men [10]. Higher rates of lower bone mass with lower T-scores have been reported in women with SCI compared to women with other disabilities [11]. Because a large proportion of spinal cord injuries occur before obtaining the peak bone mass and because the rate of bone resorption and formation is reduced, particularly below the level of injury, low values of BMD and increased risk of fracture in people with impaired spinal cord is not surprising [11]. Studies in paraplegics already found that age at injury can affect bone loss [6]. Another reason is that the term "osteoporosis" below the level of injury must be used with caution especially in quadriplegia, paraplegia and/or equivalent diseases. This concept is supported by the maintenance of bone in the spine in regions below the level of lesion because of weight bearing in seated position (i.e., in a wheelchair), and compressive stress of the fusion materials usually used following injury in the injured area (i.e., in traumatic spinal cord injury paraplegia). Force exerted by orthopaedic materials in the surrounding muscles may affect BMD of the spine, but unfortunately there are no studies to support this hypothesis. Because of the unique and individually based approach needed in the management of each disabled subject with spinal cord lesion and their complications according to bone the new term spinal cord injury related bone impairment, (SCI-related BI) is proposed as correct. In some books we found also the term bone disorder. The term bone impairment is more appropriate compared with bone disorder because includes terminology from rehabilitation science a medical specialty which interferes with all complications of spinal cord injured and follows these subjects during aging with paralysis. It is

not used here for the first time. Very experienced researchers who published pioneer papers in this field have chosen to use the term bone impairment to describe "osteoporosis" in SCI [5]. That term is also found in other conditions [12]. As traditionally used in rehabilitation, impairment refers to a problem with a structure or organ of the body, in this case bone's structure [13].

References

1. Prevention and management of osteoporosis. World Health Organ Tech Rep Ser 921: 1-164.
2. NIH Consensus Development Panel on Osteoporosis. JAMA 285: 785-795.
3. Assessment of fracture risk and its implication to screening for postmenopausal osteoporosis. World Health Organ Tech Rep Ser 843: 1-129.
4. Sambrook P, Schrieber L, Taylor T, Elli (2001) The musculoskeletal system. Edinburgh: Churchill Livingstone.
5. Garland DE, Adkins RH, Stewart CA (2013) Bone Impairment and Spinal Cord Injury. In: JH Stone, M Blouin, editors. International Encyclopedia of Rehabilitation.
6. Dionyssiotis Y (2011) Spinal cord injury-related bone impairment and fractures: an update on epidemiology and physiopathological mechanisms. J Musculoskelet Neuronal Interact 11: 257-265.
7. Frost HM (1997) Defining osteopenias and osteoporoses: another view (with insights from a new paradigm). Bone 20: 385-391.
8. Lazo MG, Shirazi P, Sam M, Giobbie-Hurder A, Blacconiere MJ, et al. (2001) Osteoporosis and risk of fracture in men with spinal cord injury. Spinal Cord 39: 208-214.
9. Garland DE, Foulkes GD, Adkins RH, Stewart CA, Yakura JS (1994) Regional osteoporosis following incomplete spinal cord injury. Contemporary Orthopaedics 28: 134-139.
10. Coupaud S, McLean AN, Allan DB (2009) Role of peripheral quantitative computed tomography in identifying disuse osteoporosis in paraplegia. Skeletal Radiol 38: 989-995.
11. Smeltzer SC, Zimmerman V, Capriotti T (2005) Osteoporosis risk and low bone mineral density in women with physical disabilities. Arch Phys Med Rehabil 86: 582-586.
12. Roato I, Porta F, Mussa A, D'Amico L, Fiore L, et al. (2010) Bone impairment in phenylketonuria is characterized by circulating osteoclast precursors and activated T cell increase. PLoS One 5: e14167.
13. http://www.who.int/classifications/icf/en/