

Bone Health Following Spinal Cord Injury

1.0 Executive Summary

There is overwhelming evidence that supports the importance of addressing bone health issues after spinal cord injury (SCI). Preserving bone mass and maintaining bone architecture are crucial to decrease an individual's future risk of developing a fragility fracture.

Much of what we know about sublesional osteoporosis (SLOP) comes from published data about men with motor complete paraplegia of traumatic etiology.

Sublesional osteoporosis (SLOP) is a condition that causes the weakening of bones in persons with SCI. It is characterized by excessive bone resorption, decreasing bone quality and an increased risk of lower extremity fragility fracture.

A higher incidence of knee region fractures exists in people who sustain SCI and the majority of fractures occur following transfers or activities that involve minimal or no trauma ([Comarr et al. 1962](#); [Ragnarsson & Sell; 1981](#); [Freehafer 1995](#)). The distal femur and proximal tibia (knee region) are typically most at risk.

1.1 What bone health issues occur after SCI?

Within the first few days following SCI, there is an increase in excreted calcium (known as hypercalciuria) that is 2-4 times that of people without SCI who are confined to prolonged bed rest. Hypercalciuria results in excessive bone resorption or breakdown ([Bauman & Spungen 2001](#)).

Longitudinal studies showed a higher rate of hypercalcemia (excessive calcium in the blood) for people with acute SCI. This effect is more rapid in the first 4-6 months post-injury and usually slows for the remainder of the first-year post-injury ([Hancock et al. 1979](#); [Frey-Rindova et al. 2000](#)).

Early studies also suggested that bone mineral density (BMD) stabilizes by 1-2 years after SCI at 25-50% below that of non-disabled peers in the hip and knee regions ([Griffiths et al. 1976](#); [Hancock et al. 1979](#); [Garland et al. 1992](#)). More recent studies suggest a continual decline in BMD implying that a new lower extremity bone mineral homeostasis is not established in the chronic stage of injury ([Demirel et al. 1998](#); [Bauman et al. 1999](#); [Eser et al. 2005](#)).

Sublesional osteoporosis develops after SCI in response to several changes in the hormonal, vascular, inflammatory, neuromuscular, and autonomic nervous systems following SCI. In addition, changes to nutritional habits, weight-bearing and the amount of daily physical activity with ageing likely also contribute to SLOP severity. The relative contributions of each factor are not well delineated.

The declines in bone mass and changes in bone quality after SCI make people more susceptible to lower extremity fractures in response to seemingly minor stresses during day-to-day activities (i.e. fragility fracture) where the leg bones are twisted during a transfer or suddenly hyper flexed in a low velocity all from a wheelchair. About 1-4% of people with SCI will get a fracture in any given year ([Zehnder et al. 2004a](#)) typically affecting the femur (thigh bone above the knee) and the tibia (shin bone below the knee).

Fragility fracture: A fracture occurring spontaneously or following minor trauma such as a fall from standing height or less. ([Kanis et al. 2001](#); [Bessette et al. 2008](#))

Fragility fractures in persons with SCI are important to prevent because they can affect mobility, the ability to function in daily life, and can be a major cause of medical expenses. Fractures after SCI do not always heal and they may be associated with other complications of immobilization such as deep vein thrombosis, pressure injuries or cellulitis.

1.2 What are the risk factors for SLOP?

There are several risk factors for developing low bone mass after SCI, including female gender, paraplegia, motor complete injury of traumatic etiology and the duration of SCI.

Clinicians and patients should be most concerned about fracture risk, not just low hip and or knee region bone density. A treating clinician can readily identify patients with a high risk of fragility fracture (5 risk factors or ****The big 2****) by completing the checklist on the right. Of these fracture risk factors, a prior fragility fracture and a low knee region BMD below the fracture threshold are the most potent predictors of future fracture (****The big 2****).

Lower Extremity Fragility Fracture Risk Factor Checklist after SCI

([Craven et al. 2008](#); [Craven et al. 2009](#); [Cervinka et al. 2017](#))

- Age at Injury < 16 years
- Alcohol Intake > 5 servings/day
- Body Mass Index < 19
- Duration of SCI ≥ 10 years
- Woman
- Motor Complete (AIS A-B)
- Paraplegia
- Prior fragility fracture*****
- Family history of fracture
- Anticonvulsant use (i.e. - Tegretol, , Gabapentin -
- Spasticity Medication
- Heparin use
- Opioid analgesia use (≥28 mg morphine for a 3 months period)
- SSRI**
- PPI**
- Knee region BMD below the fracture threshold*****
- ** "The big 2" ****

1.3 What are management options for bone problems?

Screening for secondary causes of osteoporosis, ensuring an adequate but not excessive dietary calcium and vitamin D intake, and completing a fracture risk factor assessment are important first steps.

Measuring and monitoring hip and knee region BMD should begin early following SCI, given the significant declines in hip and knee region bone mass in the first year after injury and the associated lifetime increase in fracture risk.

Biomarkers (urine, blood) provide clinical insight into the metabolic activity of bone, but imaging techniques (e.g. Dual-Energy X-ray Absorptiometry or DXA) provide insight into bone density, quality, and architecture. Conventional tools for predicting fracture risk, such as the Fracture Risk Assessment Tool (FRAX) are only valid among postmenopausal women or men 50 years of age and older.

Pharmacological Options

A number of bisphosphonates have been researched to prevent BMD decline associated with SLOP in persons with SCI. There is Level 1 (Strong) evidence for the prevention and treatment of BMD decline using bisphosphonates (clodronate, etidronate, zoledronate for prevention, and alendronate for treatment) may slow the decline of hip and knee BMD early after SCI or maintain BMD of the hip region in the chronic stage after injury.

Non-Pharmacological Options

There is a lack of evidence supporting rehabilitation interventions for the treatment of SLOP after SCI, except neuromuscular electrical stimulation (NMES) and functional electrical stimulation for cycling (FES-cycling), which can maintain or increase BMD over the stimulated areas for as long as the individual persists with the intervention.

Previously, a decision guide has been published for rehabilitation professionals on the identification and management of bone health-related issues for people with SCI ([Craven et al. 2008](#), [Craven et al. 2009](#)).