Commentary

Osteoporosis in Complete Spinal Cord Injured Persons

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Introduction

Osteoporosis induced by spinal cord injury (SCI) remains poorly understood including inducing mechanisms. Some knowledge was achieved by biopsy of the pelvic crest for histomorphic interpretation which proved to be a poor site to study (smaller amounts of bone loss). Blood and urine biochemical studies provided some insight into negative calcium, phosphorous and nitrogen balances, but failed to detect locations of loss. The advent of dual photon absorptiometry (DPA), which measured total bone mineral (TBM), and duel energy x-ray absorptiometry (DEXA), which measured bone mineral density (BMD), permitted specific site, duration and amount of bone loss measurements. However, most studies lack large populations which cause researchers to mix populations to gain numbers such as gender, age, duration of injury (DOI), neurological extent and level, and other modalities such as medications, smoking, drinking and coffee. This only taints the data and causes conflicting reports and confusion.

Most of our studies were a homogeneous young complete SCI male, under 40 years of age, population to lessen confounding although we studied other groups for comparison but without the power. Our internal studies remained consistent over a twenty year period with different methods with some findings differing from the literature. What follows is what we "think" we might know, not what we claim clearly or nearly to know.

Our first study, now a minor classic, involved a DPA total body scan of complete, both paraplegic and quadriplegic, males under 40 years [1]. Three populations were broken down into 4 groups: group I - able bodied, aged match; group II - SCI an average of 114 days after injury; group III - (same patients in group II) at 468 days after injury; group IIII - new SCI population greater than 5 years after injury (chronic). All areas of the body lost bone except the skull. The upper extremities (UE) lost bone even in paraplegics but especially in the group II compared to the group I which then slowed in the paraplegics but continued in quadriplegics. The trunk initially lost bone but

eventually gained bone. This study contradicts many papers which continue to call SCI bone loss "sublesional" osteoporosis which we think is a misnomer and believe there is a systemic component. The area of the spinal bone loss and gain remains controversial as well but our future studies would consistently find that not only does the lumbar spine loose bone early, it may gain bone mass to near able body matches controls with time. We think the gain above the pelvis may be the result from the body's mechanisms to prevent bone loss which those areas can appreciate but the lower extremities (LE) cannot.

LE studies demonstrated the most dramatic findings. Most of the bone loss was in the LE and the loss progressed as the study area moved distally. The overall LE bone loss was stabilizing in group IIII but the area that seemed responsible for this was above the knee while the area below the knee continued to loose bone. One final contribution of this paper was the concept of bone loss according to DOI. We would loosely define: acute bone loss - first 4 months; subacute loss - 5 months up to 2 years; chronic loss - after the two year period. This study would become the basis for over twenty years of SCI research. Since most fractures occurred in the LE, we would spend most of out efforts studying that area, pathologic fractures and fracture threshold [2,3].

The acute loss phase (response to injury) begins immediately after SCI and lasts approximately 4 months. The BMD "weekly" changes are: (-) 0.5% for the lumbar spine; (-) 2% at the pelvis, os calcis and entire LE (TBM); (-) .75 % at the hip; (-) 1% at the knee, both the distal femur and proximal tibia. It should be noted that studies of bone resorption markers are elevated at this time in the urine and serum and peak at twenty four weeks; bone building markers are either minimally or slightly elevated. The phase is largely osteoclastic with negligible osteoblastic effects.

The subacute phase (adaptation and adjustment) persists twelve months or longer. BMD "monthly" changes (as opposed to acute "weekly") are: (-) 2% at the pelvis, os calcis, and LE (TBM); (-) .75% at the hip; (-) 1% at the knee. Many of the bone resorption markers

have or are returning to normal. Endocrine markers such a parathyroid (PTH) may become elevated at the beginning of this phase in response to acute phase changes, but are returning to baseline by the end of this phase.

The chronic phase begins approximately two years after injury. The lumbar spine has stopped losing bone and a period of stability ensues before it begins to gain bone In our younger population it would be rare for degenerative changes to occur giving false positive information. BMD changes are: (-) 1% at LE (TBM) although some may have no and a slight increase; the hip area is relatively stable; (-) 1% annually at the knee and os calcis. BMD at the knee may show no yearly change or even some non significant increase in bone but the overall trend is lower.

Summarily, bone loss progressively accelerates from the hip to the os calcis. The lumbar spine looses a small amount of bone, approximately (-) 10%, but may eventually gain bone close to age matched able bodied. The hips decreases approximately (-) 20-25% during the acute and subacute phase (near fracture threshold) and then stabilizes but may have mild waxing and waining of bone loss. The knee looses (-) 25-30% in men and (-) 30-40% in women during the acute and subacute phase and then slows to around (-) 1% annually. The annual BMD loss of (-) 1% continues in women and tetraplegic males but some male paraplegics may see an occasional gain bone.

Many fracture studies have been hospital based. This has led to a bias that most fractures occur at the knee, the so called "paraplegia fracture", with some at the hip. This merely reflects fracture types that are most commonly admitted to the hospital. Our study of 146 consecutive pathologic fractures in 106 SCI individuals demonstrated significantly different fracture locations from the literature [4]. Locations were: UE - 10; peritrochanteric - 21; femoral shaft - 10; distal femur - 26; proximal tibia - 19; tibial shaft - 15; distal tibia - 19; ankle area - 21; foot-5 Regions of fractures were; hip area - 21 (15%); femur - 57 (39%); the knee area - 45 (31%); tibia - 74 (51%); ankle area - 40 (27%).

This study is clinical confirmation of the above bone loss studies, namely, bone loss progresses from hip to os calcis. The tibia sustains the most fractures but the knee remains the most common region of fracture which is also related to the mechanism of injury - the knee sustaining the majority of the torsional forces in transfers and falls. Lastly, there were no spine fractures in this series. The author has only seen one pathological spine fracture in a complete tetraplegic female older than 65 years of age with multiple medical problems. This may support our findings of negligible bone loss in the lumbar spine.

The fracture threshold is established for the able bodied population and defined as the BMD at a specific site below which fractures occur. BMD fracture breakpoints are the level at which the majority of fractures occur. The densitometric diagnosis of osteoporosis according to the criteria of the World Health Organization was not available when our studies began nor was there information regarding diagnosis of osteoporosis at the knee. Consequently, we dealt mainly in the percentages of bone loss compared to the able bodied as well as the percentage of bone loss when fractures occurred. We also do not know if the fracture threshold for any given fracture site for SCI is the same as the able body. Certainly, the amount of energy is less concerning our pathologic fractures.

We studied the knee BMD is 168 complete patients: one group with LE fractures and the other as controls [5]. The knee fracture threshold was found to be 0.78 g/cm2 (36% loss) with a breakpoint of .49 g/cm2 (57% loss). The knee BMD was then used as a proxy for all LE fractures: LE fractures began to occur when the knee BMD was .86 g/cm2 (25% loss); the fracture breakpoint when most LE fractures occurred was .49 g/cm2 (57% loss). Since the amount of bone loss at the knee in the acute and subacute phase of SCI is 25-30% for men and 30-40% for women, we can conclude that fracture threshold is reached by the beginning of the third year of injury(start of chronic phase) in many people with complete lesions.

We evaluate osteoporosis at the bedside or office and guesstimate their knee BMD for general information purposes. We use the

following formula and calculations to estimate bone loss at the knee (for example, a complete male 20 years after injury): acute loss - 1% weekly x 16 weeks = 16% ; subacute loss - 1% monthly x 1 year = 12%; chronic loss - 1% annually x 18 = 18%. We now add the numbers (16+12+18= 46) and estimate the person's lost 46% of their knee BMD and is between fracture threshold and breakpoint. We have compared this "estimated" BMD to the patient's "actual" knee BMD and found it to be 80% correct. It permits a general discussion for the patient and allows direction towards further evaluation and possible treatment protocols.

Treatments have not been successful to date. It seems that treatment should be initiated as soon as possible after the injury but our present armamentarium seems inadequate in preventing such a rapid bone loss. What about late treatment? The answer is also not known. All is not lost since present treatment of osteoporotic fractures in the able body have large decreases in fracture incidence, up to 50%, while only small improvements in their BMD. Perhaps normalizing BMD in our population is not so important and not the primary end point, but fracture reduction is. This concept suggests that we could treat the chronic person and expect a decrease in fracture incidence without changing the BMD.

This concept is vital. Using BMD as an endpoint may not be meaningful since increasing BMD 10%, for example, still leaves the majority of chronic patients below fracture threshold. A comparison study of no treatment versus treatment in chronic SCI who have sustained fractures would answer this query.

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