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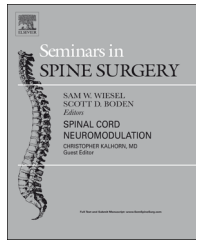

Medical management of bone loss from a spinal surgeon's perspective

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ABSTRACT

Osteoporosis is a common metabolic disorder in which the mineral and structural properties of the bone are compromised as a result of multiple, often endocrine, factors. Osteoporosis and other bone loss disorders will impact all spine surgeons. With the aging of the population, surgeons caring for adults will no doubt treat patients sustaining spinal fragility fractures from the dens to the sacrum. Moreover, all spine surgeons, even pediatric deformity specialists, will encounter challenges in successful spinal fixation related to poor bone quality. To adequately care for these patients, the physician requires a reasonable familiarity with the disease state and its implications on the spine. The surgeon must be able to identify at-risk patients, assess their bone mineral density, and, increasingly, initiate treatment.

1. Introduction

In this article, I have been tasked to "explain to a spine surgeon what they need to know about osteoporosis." First, we need to agree on a definition. The National Osteoporosis Foundation defines osteoporosis as "a systemic skeletal disease characterized by decreasing bone mass and microarchitectural deterioration of bone tissue that leads to an increased risk for bone fragility and fracture."

This disorder, and other bone loss states, demand the attention of spine surgeons in two realms. First, these patients are susceptible to major spinal injury from low-energy mechanisms. Vertebral compression fractures are among the most common stigmata of osteoporosis, but highly morbid cervical spine and other fractures may also result. Second, bone loss both contributes to the pathogenesis and complicates the treatment of other spine disorders.[1] For example, spinal deformity, especially kyphosis, progresses more rapidly in patients with poor bone quality. Then, surgical management is frustrated by the limited fixation of standard spinal implants in osteoporotic bone. Other bone loss states include Paget's Disease, osteomalacia, and secondary osteoporosis with similar, but not identical effects on the spine. Of these, secondary osteoporosis, seen in inflammatory arthritis, renal disease, hyperparathyroidism, and from a number iatrogenic causes are the most common.

This is another, quantitative definition for osteoporosis: bone mineral density below 0.75 g/cm. Normal bone contains more than 0.9 g/cm. The term osteopenia is typically used to refer to borderline bone quality with BMD 0.75–0.9 g/cm. However, osteoporosis affects both the organic and inorganic phases of bone. Recall that the organic phase provides about 40% of the dry weight of bone and is composed of several proteins, of which collagen represents 90%. This phase is responsible for bone's tensile strength. The inorganic phase is predominantly comprised of calcium hydroxyapatite, which confers bone's compressive strength.

Given our space constraints and the other papers in this special issue, I sought to provide succinct answers to several key questions about bone loss. The first of these: "Why Should I Care?" I will attempt to answer here. The others: "What Should I know?" "How do I assess?"; and "What do I prescribe?" are discussed in the sections that follow.

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As noted in other papers in this special issue, osteoporosis is common and the percentage of patients with bone loss is increasing exponentially. This increase reflects not only the aging population, but also the increased survival of patients on dialysis, after organ transplant, with cancer, and so on. Of the estimated 200 million people with osteoporosis worldwide, 54 million live in the United States. Wright et al.[2] recently estimates that, among U.S. adults over the age of 50, 43.9% had low bone mass and 10.3% had frank osteoporosis. While the largest group was, as expected, non-Hispanic white women, "a substantial number of men and women from other racial/ethnic groups also had osteoporotic BMD or low bone mass." By 2025, this burgeoning at-risk population will lead to $25 billion per year economic impact of osteoporosis on the U.S. health care system with similar burdens reported elsewhere.[3]

As a spine surgeon, you *can and must* have an impact. Even occasional interactions can identify occult disease, amplify messaging from other providers, and ultimately reverse the vicious spiral that engulfs many patents after their first fracture. Beyond that, fragility fractures of the spinal column are often one of the first manifestations of bone loss. Given that the first fracture is an excellent predictor of subsequent fractures, the spine surgeon is in an ideal position to frame the discussion in a meaningful way.[4] Unfortunately, in one large cohort survey, only 2.8% of U.S. women with osteoporotic fractures underwent densitometry testing and only 22.9% were offered management of their underlying bone loss.[5]

Increasingly, with value-based health care purchasing, your income and online quality ratings will require you to address bone quality as means of reducing postoperative complications, revisions and low value interventions. One common quality measure for providers is the rate of compliance with current Healthcare Effectiveness Data and Information Set (HEDIS) measures for management of osteoporosis after fracture: "The percentage of women 67 years of age and older who suffered a fracture and who had either a bone mineral density (BMD) test or prescription for a drug to treat or prevent osteoporosis in the six months after the fracture."[6]

2. What should I know?

While a detailed understanding of the pathophysiology of osteoporosis is not required of the typical spine surgeon (and is beyond the scope of this review), there are some aspects of osteoporosis that must be understood.

When it comes to fracture risk and the impact of bone loss of spinal stabilization, it is incumbent on the treating physician to understand that the spectrum of bone loss will have markedly different impacts on risk. That is, severe bone loss may preclude successful fixation entirely and mild bone loss might not meaningfully compromise construct rigidity.[7] Where is the patient on the continuum?

At a given spinal level, most causes of bone loss will affect the cancellous bone earlier and more dramatically than the cortical bone.[8,9] Historically, type I, or postmenopausal osteoporosis, has been described as having an earlier onset and mainly trabecular bone affect. Type II, or senile, osteoporosis was found later in life, affecting both cortical and trabecular bone. Type III or secondary osteoporosis, is that associated with other disease states or medications. This group is highly diverse as the severity and type of bone loss varies with the underlying mechanism. Spine surgeons should be aware of the many conditions that risk bone loss (Table 1). Common medications include corticosteroids, some anticonvulsants, chronic heparin therapy, and chemotherapeutic agents. Frequently implicated disease states include any associated with long-term immobility (e.g., stroke), thyrotoxicosis, beta-thalassemia, and multiple myeloma.

Similarly, regional factors and the relative make-up of cortical vs. cancellous bone means that some spinal levels are more severely affected. The differential impact of bone loss on fixation to the vertebral elements will be discussed in more detail in other papers in this special issue. The higher baseline mineralization and regional biomechanical factors result in relative protection of the lumbar spine and subaxial cervical spine.[10] The highly cancellous nature of the dens and sacrum and the loading patterns to which they and the thoracolumbar junction are exposed, place them at higher risk for both fracture and fixation failure.[11,12] Issues surrounding end levels, peri-implant fractures and proximal junctional kyphosis are discussed in more detail in other papers in this special issue.

Often, a great deal of emphasis is placed on immediate resistance of osteoporotic bone to loading, whether from deforming forces that might induce fracture or from pull-out forces on spinal implants. It is important to remember that osteoporotic patients often have weaker connective tissue, slower bone healing, and ultimately weaker fracture callus and fusion mass than normal controls.[13–15] For these reasons, implant removal should be considered rarely in these patients. Additionally, fatigue resistance must be considered when designing spinal constructs.[16]

3. How do I assess?

Even if you do not plan to treat the bone loss yourself, you *must* understand what these tests mean to properly plan or defer surgery in these patients. A critical concept is that the presence of a fragility fracture is THE primary indicator of osteoporosis. Frequently, patients treated for a low-energy thoracic vertebral compression fracture will return stating that their family doctor told them they "don't have osteoporosis" based on a DXA scan t-score above −2.5. This is simply not true.

As anywhere in spine care, proper evaluation begins with the History and Physical Exam. The goal of the history is to identify the variety of risk factors for osteoporosis, listed in Table 1, that should be familiar to all spine surgeons. Often, but certainly not always, osteoporotic patients will be thin and kyphotic.[17] They should not have diffuse bone pain or tenderness. Absent complicating fractures, osteoporosis should be asymptomatic. In patients with bone pain, another disorder such as osteomalacia or lymphoma should be considered in the differential.

The utility of routine laboratory testing is debated in otherwise healthy individuals at risk for osteoporosis.