Low bone density is not always bisphosphonate deficiency

In general medical practice, low bone mineral density is generally assumed to be osteoporosis. Once vitamin D levels have been evaluated (and corrected, if low) and a few potential causes of secondary osteoporosis excluded, patients with significantly low T scores are often treated with bisphosphonates to blunt the overall osteoporotic process and reduce the likelihood of future fragility fractures.

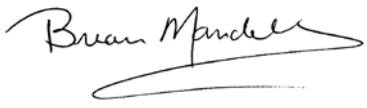
A few common conditions dramatically underscore the potential difficulties in distinguishing the hyperosteolytic biology of osteoporosis from disorders of bone hypoproduction or defective mineralization. Patients with a severe or complicated gastrointestinal malabsorptive state such as a history of gastric bypass surgery are included in this group. Management of transplantation patients may also be challenging.

But perhaps the most complicated metabolic bone patients to manage are those with severe chronic kidney disease. In this issue of the *Journal*, Dr. Paul Miller (page 715) and, in an accompanying commentary, Dr. Maria Coco (page 684) discuss the problems, some potential bone-protective strategies, and some of the controversies faced by clinicians treating bone disease in patients with chronic kidney disease.

While patients with chronic kidney disease who have low T scores are often comanaged by nephrologists and specialists in metabolic bone disease, the discussion of the pathophysiologic pathways resulting in reduced bone density is germane to many of us. A documented low T score does not equal osteoporosis and thus should not lead us to automatically prescribe a bisphosphonate.

Clues to the presence of a disease associated with secondary osteoporosis or osteomalacia should be sought in any patient with a low T score. Some of these clues are adenopathy on examination, a personal or striking family history of nephrolithiasis, unexplained anemia, thyroid disease, a low anion gap, an unexplained change in blood pressure, a particularly alkaline urine, frequent loose stools, and disturbances of phosphate or calcium.

The era of ignoring osteoporosis is fortunately coming to a close. But we should not cavalierly go where the generation of internists before us could not go—to our prescription pads. Low bone density is not a one-size-fits-all disorder. We need to carefully consider other diagnostic and therapeutic options before assuming that low bone density is due to osteoporosis in every patient. These two articles should stimulate serious thought about possible alternative diagnoses to the now frequently diagnosed "osteoporosis."



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doi:10.3949/ccjm/76a.12001

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