Fragility fractures in chronic kidney disease: A clarification of views

(DECEMBER 2009)

TO THE EDITOR: I was pleased to see my article on fragility fractures in patients with chronic kidney disease (CKD) in the *Cleveland Clinic Journal of Medicine*[1] and your preamble Letter from the Editor.[2]

However, Dr. Coco's accompanying editorial[3] misquoted a particular point I cautiously and consistently make—not only in the *CCJM* article, but in other invited papers on the topic of fractures in CKD. I specifically state that bisphosphonates should only be considered in stage 4–5 CKD in *fracturing* patients, not just those with "low bone mineral density," who have clear-cut osteoporosis by exclusion of other causes of fractures in this population. Hence, Dr. Coco's statement that "… the author advocates the use of bisphosphonate therapy in patients with chronic kidney disease who have low bone mineral density" is inaccurate.

If one carefully reads the last four paragraphs of my paper on page 721, one will see that I emphasize this caution repeatedly and even specifically state: "Treating only on the basis of low bone mineral density and other risk factors seems to be associated with greater risk than benefit."

Thank you for your consideration.

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3. **Coco M**. Treating the renal patient who has a fracture: opinion vs evidence. Cleve Clin J Med 2009; 76:684–688.

doi:10.3949/ccjm/77c:02003

IN REPLY: Bone disease in the patient with chronic kidney disease (CKD), especially in the presence of a fracture, is indeed a vexing problem. Clinically, it is very difficult to differentiate between low bone turnover—not uncommon in patients with CKD—and patients who have osteoporosis. Clinically, these patients present similarly: both can have abnormal bone density measurements (usually low bone mineral density with T scores less than –2.5 standard deviation), and both can have fractures. But both should not be treated the same without further evidence.

In Dr. Miller's article, bisphosphonate and other therapies are named as possible treatments for "osteoporosis" in patients with CKD stages 1 through 3. "Treatment decisions are more difficult … in stage 4 and especially stage 5 chronic kidney disease with fragility fractures…" (page 721).

Dr. Miller indeed states that "patients without fractures with stage 5 … should not be given bisphosphonates …" He also states, "Treating only on the basis of low bone mineral density … seems to be associated with greater risk than benefit." In Dr. Miller's opinion, the latter group of patients may be treated with a bisphosphonate if there has been a fracture. However, many of these patients may have fractured because of low turnover bone disease; unfortunately, they cannot have "clear-cut osteoporosis by exclusions of other causes." Bisphosphonate therapy may further suppress bone activity (if there is any activity left) and may predispose to extraosseous and cardiovascular calcifications and further non-bone pathology.

Dr. Miller does caution regarding unknown risks in these patients with advanced kidney disease.

Treating metabolic bone disease is certainly not straightforward, especially when present in the fracturing renal patient. We need more evidence before making treatment paradigms.

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doi:10.3949/ccjm/77c:02004