Kidney stones

(OCTOBER 2009)

TO THE EDITOR: Thanks for the excellent review articles on nephrolithiasis in your October 2009 issue.[1,2]

Dr. Hall[1] cites studies in which patients given the alpha blocker tamsulosin (Flomax) or the calcium channel blocker nifedipine (Procardia) had improved rates of kidney stone passage compared with placebo. As a primary care physician, I am often confronted with the challenge of managing a patient who is waiting for a kidney stone to pass while taking tamsulosin. Is Dr. Hall aware of any clinical studies, or at least theoretical reasons, which would support adding nifedipine in such cases?

Secondly, Dr. Hall cites studies that demonstrated that a higher intake of dietary calcium is actually associated with fewer calcium stone events in both men and women. An unanswered question is whether patients taking calcium supplements for osteoporosis or osteopenia can safely continue to do so after a calcium stone event, or indeed, whether calcium supplementation might actually be helpful in preventing a recurrent calcum stone.

If there is an absence of randomized studies to answer these questions, Dr. Hall's recommendations based on his expert experience would be most welcome.

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REFERENCES

1. **Hall PM**. Nephrolithiasis: treatment, causes, and prevention. Cleve Clin J Med 2009; 76:583–591.
2. **Samplaski MK, Irwin BH, Desai M**. Less-invasive ways to remove stones from the kidneys and ureters. Cleve Clin J Med 2009; 76:592–598.

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IN REPLY: I thank Dr. Keller for his kind letter.

With respect to expulsive therapy, Dellabella et al[1] randomly assigned 210 patients to receive nifedipine, tamsulosin, or phloroglucinol. All the patients also received a corticosteroid. The most effective therapy was tamsulosin, though this was not a placebo-controlled study. In a separate study, Borghi et al[2] compared methylprednisolone plus nifedipine and methylprednisolone plus placebo. The nifedipine-methylpednisolone combination seemed to result in more prompt stone passage.

With respect to calcium supplements in calcium kidney stone disease, Curhan et al[3] prospectively examined stone risk associated with dietary calcium as well as calcium supplements. This seemed to show that with calcium supplements there was no increased risk, and there may have even been some benefit. In another study by Borghi et al,[4] normal dietary calcium intake was shown to be associated with lower stone risk than a low calcium intake. Further, the study by Curhan et al[3] seemed to indicate the same.

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REFERENCES

1. **Dellabella M, Milanese G, Muzzonigro G**. Randomized trial of the efficacy of tamsulosin, nifedipine and phloroglucinol in medical expulsive therapy for distal ureteral calculi. J Urol 2005; 174:167–172.
2. **Borghi L, Meschi T, Amato F, et al**. Nifedipine and methylprednisolone in facilitating ureteral stone passage: a randomized, double blind, placebo-controlled study. J Urol 1994; 152:1095–1098.
3. **Curhan GC, Willett WC, Knight EL, Stampfer MJ**. Dietary factors and the risk of incident kidney stones in younger women: Nurses' Health Study II. Arch Intern Med 2004; 164:885–891.
4. **Borghi L, Schianchi T, Meschi T, et al**. Comparison of two diets for the prevention of recurrent stones in idiopathic hypercalciuria. N Engl J Med 2002; 346:77–84.

doi:10.3949/ccjm/77c.02002

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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REFERENCES

1. **Miller PD**. Fragility fractures in chronic kidney disease: an opinion-based approach. Cleve Clin J Med 2009; 76:715–723.
2. **Mandell BF**. Low bone density is not always bisphosphonate deficiency (From the Editor). Cleve Clin J Med 2009; 76:683.
3. **Coco M**. Treating the renal patient who has a fracture: opinion vs evidence. Cleve Clin J Med 2009; 76:684–688.

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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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