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The rationale for calcium and vitamin D supplementation in the prevention and treatment of osteoporosis is that low dietary calcium intake and/or vitamin D deficiency or insufficiency may contribute to bone loss.[1] Additionally, supplementation is generally recommended as an adjunct to other osteoporosis therapies (antiresorptive and anabolic agents, and strontium ranelate).

Nevertheless, it is still unclear whether the addition of calcium/vitamin D supplements leads to an incremental benefit in patients taking these bone-active drugs. It is also unclear as to what extent osteoporosis treatment maintains its efficacy in patients with an inappropriate intake of calcium or with vitamin D deficiency.

Patients receiving treatment for osteoporosis in a routine clinical setting are often older and frailer than those recruited in clinical trials. Thus, individuals placed on pharmacological treatment for osteoporosis are likely to be at the highest risk of vitamin D deficiency.[2] This high proportion of patients on pharmacological treatment for osteoporosis with vitamin D deficiency may sound somewhat surprising, since all guidelines recommend that any pharmacological intervention should include calcium and vitamin D supplements. Implementation of these guidelines, however, is hampered by a number of factors.

Recently, there have been concerns in the literature about potential risks (ie, excess cardiovascular events) with calcium supplementation and high normal serum calcium levels. Additionally, concerns have been expressed that higher treatment doses of vitamin D than those conventionally used may induce vitamin D toxicity.

The Institute of Medicine has issued guidance on vitamin D and calcium intake. Their consensus report found strong support for the use of vitamin D for bone health, but not for other conditions. Vitamin D is involved in calcium homeostasis, and calcium-associated toxicities at high concentrations include kidney and tissue damage.[3] Resolving whether the benefits outweigh the risks will determine the appropriateness of supplemental nondietary calcium in fracture prevention.

The most obvious reason to supplement a patient's anti-osteoporosis medication with calcium and vitamin D is that all clinical trials having demonstrated antifracture efficacy have been performed by adding—in both groups (placebo and treated)—a combination of calcium and vitamin D.

Additionally, it has been demonstrated that differences in vitamin D status may affect the anticatabolic response to anti-osteoporotic treatment,[4] and that optimal vitamin D repletion appears to be a prerequisite for maximizing the response to antiresorbers in terms of both bone mineral density changes and antifracture efficacy. Greater benefits can even be achieved when blood vitamin D levels are higher than those recently recommended by the Institute of Medicine to maintain bone health.[5]

In conclusion, it is evident that calcium and vitamin D supplementation should be a prerequisite for maximizing the response to anti-osteoporosis drugs in terms of both bone mineral density changes and antifracture efficacy. However, the treatment adherence to these calcium and/or vitamin D formulations is modest as a result of poor tolerability.[6] The consequence of this is that many patients receive neither calcium nor vitamin D. Given this issue, more effort may be usefully applied to encourage persistence with treatment. ■

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