

Fosamax (Alendronate Sodium)

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These days, we are bombarded with advertisements for prescription drugs. Many of our patients are taking these drugs. In 2007, total revenue for the pharmaceutical industry was more than half a trillion dollars and its profits were more than \$79 billion.¹ As chiropractors, we need to be informed about these prescription drugs so we can educate our patients about their effects, side effects and dangers, as well as suggest safer alternatives when appropriate. (Don't count on their medical doctor or pharmacist to fully inform them.)

Candidates for osteoporosis commonly are advised to get a dual-energy X-ray absorptiometry (DEXA) scan, an enhanced form of X-ray that detects bone density. The test results are reported in the form of two scores. The T score shows the amount of bone you have compared to a young adult of the same gender with optimal bone mass. A T score above -1 is considered normal. (A -1 T score represents a 10 percent loss of bone density.) A T score between -1 and -2.5 is classified as osteopenia and a score below -2.5 is defined as osteoporosis. The Z score reflects the density of your bones compared to others of your gender, age and size.

Once bone density is determined, Fosamax (alendronate sodium) often is prescribed to treat and prevent osteoporosis. Fosamax works by inhibiting osteoclastic activity. Normally, old bone is removed by osteoclasts and then replaced with new bone by osteoblasts. While Fosamax does, in fact, increase bone density, it does not particularly increase bone strength. It's similar to trying to repair an old house by nailing new boards to a rotted structure; the walls are denser, but not a whole lot stronger.

Merck & Co., Inc., the manufacturer of Fosamax, claims the drug is proven to prevent fractures. However, this is somewhat misleading. Although the Fracture Intervention Trial (a randomized, double-masked, placebo-controlled trial designed to test Fosamax's ability to reduce the rate of fractures in postmenopausal women with low hip bone marrow density) did show a reduction of fractures in patients with a T score below -2.5, there was no reduction in fractures for those who had a T score above -2.5.²

On the contrary, a recent study published in the *Journal of Bone and Joint Surgery* suggests an increase in femur fractures with long-term use of Fosamax.³ Numerous lawsuits have been filed claiming osteonecrosis of the jawbone was caused by Fosamax. And according to the *Physicians' Desk Reference*, possible side effects include abdominal pain, bone and joint pain, constipation, diarrhea, indigestion, muscle pain, nausea, abdominal distension, acid backup, difficulty swallowing, esophageal ulcers, gas, headache, vomiting, changes in taste, inflammation of the stomach, rash and skin redness.⁴

Nevertheless, Merck is not experiencing much setback. Merck's 2006 sales of Fosamax were \$30 billion. In 2007, Merck spent \$7.6 billion on marketing and administration and \$4.9 billion on research and development.⁵ The local CVS pharmacy in my town charges \$94 for a month's supply of Fosamax.

John R. Lee, MD, in his book *What Your Doctor May Not Tell You About Menopause*, makes a good case that estrogen dominance contributes to osteoporosis and estrogen dominance can be controlled with natural progesterone.⁶ Adequate weight-bearing exercise and intake of calcium and vitamin D have been shown to strengthen bones.

Hyperthyroidism has long been thought to cause bone loss. A recent study published in *Molecular Endocrinology* confirms this idea and indicates high levels of thyroid hormones do cause bone loss, but contrary to popular belief, low thyroid stimulating hormone (TSH) is not contributory to osteoporosis.⁷ Most medical doctors base the dosage of thyroid medication on TSH levels and consider a low TSH level to be indicative of hyperthyroidism, which is not necessarily true. A free T3 test, along with clinical correlation of the patient's signs and symptoms, is a much better indicator of thyroid status than a TSH test.⁸

Likewise, another study presented in *Molecular Endocrinology* indicates it's a lack of thyroid hormones (hypothyroidism) rather than elevated TSH levels that cause abnormal skeletal development.⁹ Consequently, undermedicated hypothyroid patients might be at increased risk for bone loss.

References

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