



Osteoporosis Canada

Ostéoporose Canada

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osteoporosis

update

a practical guide
for Canadian physicians

Measures of success

How to gauge your patient's
response to therapy

questions & answers

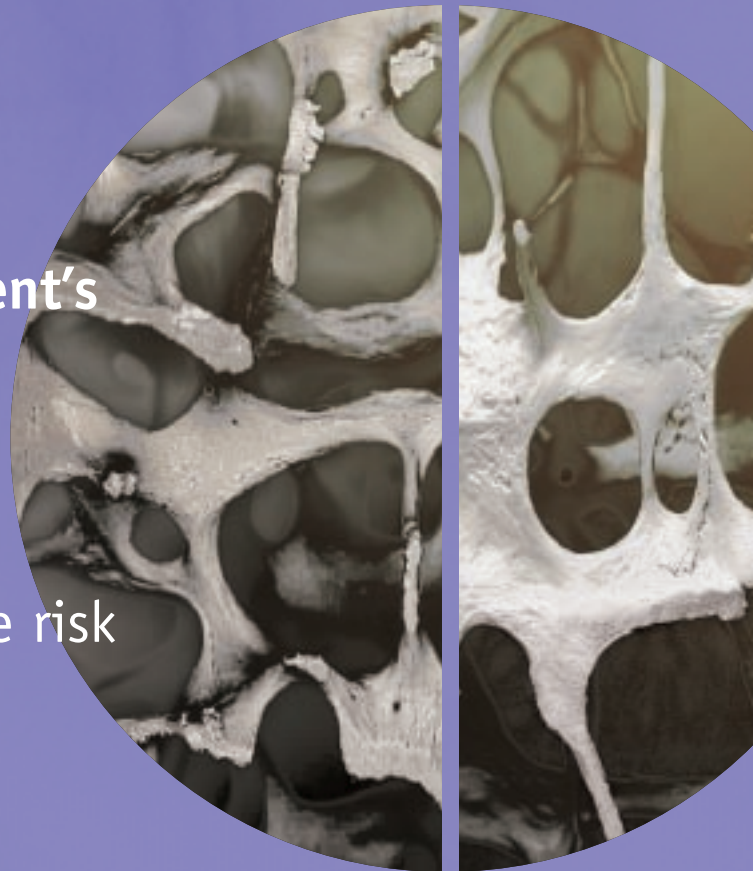
Vitamin B12 and BMD

Beta blockers and fracture risk

Managing osteoporosis
in celiac patients

case study

Are bisphosphonates
working for this woman?



Resources
& conference
details
Page 9

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What factors contribute to a successful outcome?



Louis-Georges Ste-Marie, MD, CPSQ, is an endocrinologist and Director of the metabolic bone diseases laboratory at the Centre de recherche du Centre hospitalier de l'Université de Montréal, Hôpital Saint-Luc, and Associate Professor of Medicine, Université de Montréal.

Gauging a patient's response to pharmacologic therapy is one of the challenges clinicians face in managing their patients with osteoporosis. Is a decline in BMD, or no change, a sure indication that therapy should be stopped or switched? Are new fractures while a patient is on antiresorptive medications always a sign that the primary goal of therapy, fracture reduction, has not been reached and a call for immediate action? What other possible causes implicated in treatment failure (e.g. poor adherence, calcium and vitamin D deficiency, concomitant conditions) should a physician investigate in deciding whether a change to the current therapy is warranted? What complementary information do bone turnover markers provide about the effectiveness of treatment?

This issue of *Osteoporosis Update* addresses these and other important questions. The feature article provides an overview of strategies clinicians can use to help evaluate response to treatment, even in the absence of a clear consensus on the optimal method of assessment in individual patients. It gives a useful checklist of questions they should ask to establish possible causes of non-response to therapy and suggests steps to follow when a change of therapy is indicated. In his case commentary starting on page 5, Dr. Gregory Kline examines the same subject, commenting on the specific case of a 50-year-old postmenopausal woman whose serial BMD remains unchanged from baseline after five years of bisphosphonate therapy.

In the Q&A on page 9, three experts offer insights on timely issues. Nutritionist Jennifer Dunne explains the role of vitamin B12 in bone loss and the possible risk to people who follow strict vegetarian diets; Dr. Suzanne Morin discusses the latest evidence on the effect of beta blocker use on BMD and fractures; and Dr. Tim Murray answers questions on screening and managing bone loss in patients with celiac disease, known to have a higher rate of osteoporosis than the general population.

Osteoporosis Canada is committed to providing current evidence-based information that will be useful to general and family physicians in their daily practices. Please send any questions or comments to mackinnon@parkpub.com. The Scientific Advisory Council welcomes your feedback and will be pleased to address your concerns.

Osteoporosis Canada is the new name for the only national charity serving Canadians with osteoporosis and those at risk. The name change was approved in June 2005 by the members of the former Osteoporosis Society of Canada at its Annual General Meeting, and by Industry Canada in August 2005.

Is bisphosphonate therapy working in this patient?

Patient R.M. is a 50-year-old woman with a history of mild multiple sclerosis. Aside from using a cane to walk, R.M. has no significant disability. At diagnosis 15 years ago, she received high-dose glucocorticoids for three months but has not received any further medical therapy for her MS since. She became menopausal at age 45. A bone mineral density (BMD) measurement at that time showed a lumbar spine T score of -3.4 and total hip T score of -3.2 . Her Z scores at these sites were -2.7 and -2.6 , respectively. R.M. declined estrogen replacement therapy and has now been on bisphosphonate therapy for five years. She has not had any fractures, but her repeat BMD is completely unchanged from her baseline pretreatment. Her referring doctor wonders if this constitutes a failure of therapy.

Dr. Gregory Kline comments: Failure of bisphosphonate therapy is not well defined in the literature. True resistance to the antiresorptive effects of bisphosphonates has not been described outside of the problems of non-adherence to therapy and malabsorption of the drug when taken orally. Fracture occurrence and decline of BMD (or failure to increase) are the usual events that raise doubts about whether therapy is working.

Incident fracture while on therapy is indeed a concern. Some provincial formularies regard this as evidence of drug failure and allow reimbursement for alternate antiresorptive medications. Although I am in favour of increased access to agents with differing mechanisms, it is important to recognize that this definition of “failure” is not evidence-based. While the major clinical trials of approved antifracture agents report reduced fracture risk, no drug has yet been shown to prevent all fractures. In the pivotal clinical trials involving antiresorptive therapy, the three-year vertebral fracture rate ranged from 8% to 15% over three years (Black DM et al. *Lancet* 1996;348:1535–41; Harris ST et al. *JAMA* 1999;282:1344–52; Ettinger B et al. *JAMA* 1999; 282:637–45).

Since all of these studies showed a decrease in fracture rate with these therapies compared to calcium and vitamin D alone, the occurrence of a fracture, especially in the first six months of taking an antiresorptive drug, does not clearly indicate drug failure. At the same time, these as well as the parathyroid hormone (Neer RM et al. *N Engl J Med* 2001;344:1434–41) trials showed that multiple incident fractures were distinctly unusual while on therapy. A diagnosis of “therapy failure” may be reasonable in patients who sustain two or more

fragility fractures despite adherence to one of these medications.

BMD changes are more difficult to interpret. It is beyond the scope of this commentary to discuss the controversy surrounding the relative importance of BMD change during therapy in great detail, but a few points should be noted.

- Individual patients on therapy may or may not see their BMD change in a manner identical to the “mean expected change” often reported in clinical trials.
- A range of BMD responses may occur, including no change and even a slight decrease (Sarkar S et al. *JBMR* 2002;17:1–10; Cummings SR et al. *JAMA* 2000;283:1318–21). This raises the question whether patients are getting more or less benefit depending on their individual BMD response, an issue that has been addressed in post hoc analyses of the clinical trials involving risedronate, alendronate and raloxifene (Sarkar S et al. *JBMR* 2002;17:1–10; Cummings SR et al. *JAMA* 2000; 283:1318–21; Watts N et al. *J Clin Densitom* 2004;7:255–61). The relative anti-fracture benefits appear to be maintained regardless of whether BMD increases, remains the same, or even decreases (Watts N et al. *JBMR* 2005;20:2097–104).

Lack of BMD improvement on therapy (and even small declines) are not clear evidence of therapy failure, and conversely, a person with impressive BMD gains who suffers a fracture should not necessarily be reassured.

So, why bother with follow-up BMD measurement? This is again a point of controversy — in some cases, a positive change may reinforce ongoing adherence to therapy, but most physicians probably use it to pick up the occasional case where BMD is rapidly and consistently declining, possibly indicating a secondary (undiagnosed) bone disease. Whether this is a cost-effective way of screening for secondary bone diseases is debatable, but it leads us back to our case.

Further investigations

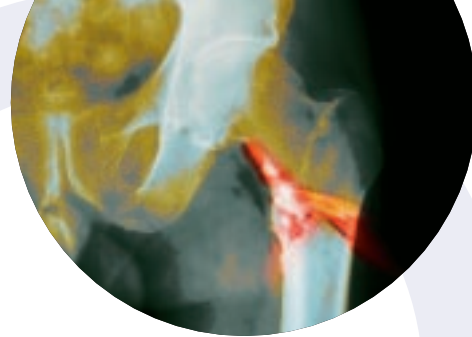
Strictly speaking, there is no evidence of therapy failure in our patient, but a “red flag” in her history warrants a second look: her extremely low T scores are inconsistent with a diagnosis of “simple” postmenopausal osteoporosis, especially since this BMD test was done within a few months of her last menses. Looking at her Z scores

Continued on page 6

Dr. Gregory Kline is an endocrinologist and Associate Medical Director of the Foothills Medical Centre. He is Clinical Assistant Professor of Medicine at the University of Calgary, in Alberta.

Non-response to therapy: a call for action?

For clinicians, early detection should be a primary concern



The overall goal of osteoporosis management is to reduce the devastating effects of the disease on a person's health and quality of life. Fracture risk assessment and prevention are the main targets of therapeutic intervention. But, at present, there is no clear consensus on the optimal method of assessing response to treatment in individual patients. Current strategies include serial measurement of bone mineral density (BMD) using dual-energy x-ray absorptiometry (DXA) and biochemical markers of bone turnover.

Manifestations of non-response

In the absence of evidence-based guidelines, the following signs in a patient on osteoporosis drug therapy should lead to further investigation:

- Any clinical fracture and clinical or morphometric vertebral fractures (remember to measure height as a marker of vertebral fracture; see *Osteoporosis Update* Winter 2005, vol. 9 no. 1 and Spring/Summer 2005, vol. 9 no. 2)
- A decrease in BMD after serial measurements
- No change in biochemical markers of bone turnover

Clinical or morphometric fractures

A new fracture, especially within the first six months of taking an antiresorptive medication, is not a sure sign of non-response to osteoporosis therapy, but it should lead to further investigation. While no treatment has been shown to reduce all fractures, studies show that multiple incident fractures are rare while a patient is on therapy.¹⁻⁴ Further, physicians should be aware that a prevalent vertebral or nonvertebral fragility fracture markedly increases the risk of a future fracture.⁵

Bone density: not the only indicator

BMD is only one of many contributors to bone strength and fracture risk reduction. Bone strength is derived from both quantity (density and size) and quality (structure, material properties and turnover). Stability or a rise in BMD is associated with fracture risk reduction for approved osteoporosis therapies, while a decline of BMD greater than the least significant change (LSC, defined below) is cause for clinical concern, because fracture risk increases as BMD decreases. But the rela-

tionship between BMD increase and fracture risk reduction is complex and nonlinear.⁶

Patients often have repeat DXA studies — usually every one to three years — to evaluate changes in BMD over time. If used correctly, serial BMD testing can be a helpful clinical tool, but DXA results should not be viewed as the only indicator for management success because therapy may or may not be associated with significant increases in BMD.⁷

In clinical trials, the majority of osteoporosis patients treated with antiresorptive or anabolic drugs stabilize or increase their BMD and benefit from a reduction in fracture risk. In clinical practice, approximately 10% of elderly patients treated with a bisphosphonate have been shown to lose BMD, defined as a BMD decrease more than the LSC at a 95% level of confidence, a value that should be calculated for each centre performing DXA scans.⁸

Bone turnover markers provide complementary information

Bone turnover markers can be used to rapidly assess adherence and effectiveness of pharmacologic interventions. They have emerged as powerful management tools for osteoporosis, since they provide information that is both different and complementary to BMD measurements.⁹ Because of the coupling between bone resorption and formation in the remodelling cycle, markers of bone resorption (within one to three months) as well as formation (within three to six months) will fall or rise in parallel in response to antiresorptive and anabolic drug therapies.

Most currently approved osteoporosis therapies are antiresorptive and produce a rapid reduction of bone turnover that reaches its nadir in three to six months, followed by a plateau. For the clinician, the primary concern is the early identification of non-responders, i.e. of patients who fail to demonstrate the expected decrease in bone remodelling and therefore, the expected reduction in fracture risk. The optimal threshold of bone marker change that will lead to the maximal fracture reduction is yet to be defined. Further research is needed in large clinical trials of each therapeutic regimen to establish the cut-offs of each bone turnover marker based on the probability of fracture.

EXPLORING THE CAUSES

To further investigate before making any changes to the prescribed drug therapy, the clinician should exclude all possible causes of non-response, including poor adherence to treatment, calcium or vitamin D deficiency, malabsorption due to undiagnosed comorbidities, and BMD measurement errors. The following checklist may provide useful clinical questions.

1. Is your patient taking the **MEDICATION REGULARLY** and **AS DIRECTED**?

Randomized clinical trials (RCTs) have shown an adherence to antiresorptive therapies of 75% to 90%.¹⁰ In a clinical practice setting, however, the rate is significantly lower. A large cohort study on alendronate therapy adherence drawn from a US retail pharmacy database population showed an adherence to drug therapy of 30% (alendronate daily) and less than 50% (alendronate weekly). The lowest rate was observed in patients who had recently started bisphosphonate therapy (25.5% on weekly, 13.2% on daily regimen, $p < 0.001$).¹¹ The patient should understand the dosing and dosing intervals and that incorrect administration of antiresorptive drugs can make them less effective. Monitoring may improve patient adherence to therapy considerably. A recent RCT on raloxifene in the UK showed that following up and presenting patients with information on response to therapy increased adherence by 57% at one year.¹²

2. Is your patient taking **CALCIUM** and **VITAMIN D** supplements?

Calcium and vitamin D therapy have demonstrated anti-fracture efficacy. Although it might not be sufficient as the sole therapy for osteoporosis, routine supplementation with calcium (1500 mg/day for people over age 50) and vitamin D₃ (800 IU/day) is still recommended as adjunct therapy to the main pharmacologic interventions with antiresorptive and anabolic drugs.^{13,14} Osteoporosis prevention and treatment require adequate vitamin D intake.

3. Does your patient present with any **CONDITIONS** leading to **MALABSORPTION** of the drug?

The following conditions may impair absorption of antiresorptive drugs:

- Gastric motility disorder (diabetes)
- Factors reducing calcium absorption (calcium carbonate supplement not taken with meal, achlorhydria)
- GI causes such as Crohn's, celiac disease. The prevalence of celiac disease in the US is 1:133 and even higher in people with osteoporosis.^{15,16}

4. Are the **SERIAL MEASUREMENT BMD** scores **ACCURATE**?

Measurement error must be considered when evaluating serial assessments. A clear understanding of how to interpret serial measurements is necessary to determine whether a change is real and not random fluctuation or artefact. Whenever possible, the patient's initial and follow-up scans should be done on the same instrument, using the same procedure. If comparison between instruments is required, e.g. after densitometer replacement, a formal cross-calibration of the instruments is necessary before attempting to compare results.¹⁷

If the patient shows a **decrease in BMD**, analyze whether the change is real. In order to be confident (at 95% confidence intervals) that the change in BMD is a true change, it must be greater than the LSC for the site being measured (about 5% for the spine and total hip and 7% for the femoral neck). In other words, a patient must lose more than this amount for a change to be significant.

Therapy should not necessarily be stopped or dismissed as unsuccessful based on BMD changes alone.¹⁸ Some postmenopausal women continue to experience BMD loss during the first year of antiresorptive therapy, and those with the greatest drops are most likely to gain BMD later on if they continue therapy. Treatment should be maintained in patients whose BMD decreases initially, because it will usually increase with continued treatment, ending in an overall gain. Also, patients who gain large amounts of bone in the first year and lose in the second year are not necessarily failing therapy — this phenomenon may indicate a correction to a random error that occurred in the earlier BMD change (termed regression to the mean).¹⁹

Next steps

After ruling out all these possible causes, consider the following strategies:

- Switch to a bisphosphonate if the patient is taking estrogen, calcitonin or raloxifene.
- Change to another bisphosphonate if the person is already on bisphosphonate therapy.
- Try another drug delivery system (IV route).
- Consider combination therapy.
- Refer to a speciality clinic.
- Switch to teriparatide. ●

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Continued from page 3

(which describe BMD in reference to an age-matched average, unlike the T score which compares to a young female average) provides a clue. A Z score less than –2 or –2.5 raises suspicion that a patient's BMD is even lower than would be expected. In R.M.'s case, this warning sign was recognized upon initial presentation at age 45 but attributed to the glucocorticoid use 10 years earlier. In retrospect, a three-month exposure to prednisone was probably insufficient to explain these very low BMD results.

Upon further questioning, the patient was in good health and not taking any medications known to interfere with bone metabolism. Other than self-diagnosed lactose intolerance, she had no symptoms. A physical exam was negative except for the fact that she was of thin build, with a BMI of 17.5 kg/m².

Additional investigations showed a normal serum calcium, phosphate, alkaline phosphatase, TSH and creatinine. Hemogram showed a very mild anemia but was otherwise normal. Her vitamin D (25-OH vitamin D) level, however, was moderately low at 29.0 mmol/l (levels above 40 mmol/l are considered normal, but for optimal bone health, we look for levels above 75 mmol/l). Subsequent studies showed strongly positive endomysial antibodies and a diagnosis of celiac disease was later proven.

Routine screening for secondary bone disease (CBC, creatinine, calcium, phosphate and alkaline phosphatase and possibly protein electrophoresis) is commonly recommended, although its cost effectiveness has been questioned, at least in the evaluation of apparent postmenopausal osteoporosis (Jamal SA et al. *Osteoporos Int*

2005;16:534–40). However, reliance upon the detection of rapidly declining BMD is probably also not a great strategy. In this case, the very low T and Z scores would likely have justified a more aggressive investigation at the start.

Common causes of secondary osteoporosis detected by lab investigation include hyperparathyroidism, hyperthyroidism and myeloma, although these are far less common than secondary osteoporosis detected by history (glucocorticoid or alcohol use etc.). We increasingly recognize vitamin D deficiency as a contributor to low BMD and fracture, and selective screening for deficiency by measuring 25-hydroxy vitamin D may be useful. As well, gastrointestinal ailments such as celiac disease, inflammatory bowel disease and post gastropasty malabsorption are also important to consider. One recent paper noted a marked increase in the prevalence of celiac disease among people being assessed for low BMD (Stenson WF et al. *Arch Intern Med* 2005;165:393–9). This patient probably did have symptoms of celiac disease, but they were misinterpreted as “lactose intolerance.” The mechanisms of bone loss in such patients are multifactorial and include malabsorption of vitamin D and other micronutrients.

Case resolution

Intestinal biopsy confirmed celiac disease and R.M. was instructed to follow a gluten-free diet. She stopped bisphosphonate therapy and two years later, her BMD had increased by 8% at the lumbar spine and she remained fracture-free. Further consideration of anti-resorptive therapy was made based upon a reevaluation of her fracture risks at that time. ●

q.

Is vitamin B12 important for maintaining bone mineral density (BMD)? Does a balanced diet provide enough, or are supplements recommended? Is there any evidence that people on vegetarian, raw food diets are more at risk of osteoporosis?

Jennifer Dunne, RD, replies: Vitamin B12 has recently been implicated as a nutrient that may affect BMD. There are several reasons for this:

- People with pernicious anemia may have an increased risk for osteoporosis and bone fractures (Eastell R, Vieira NE, Yergey AI et al. *Clin Sci* 1992;82:681–5; Goerss JB, Kim CH, Atkinson EJ et al. *J Bone Miner Res* 1992;7:573–9; Melton ME, Kochman ML. *Metabolism* 1994;43:468–69).
- Osteoporosis is also common in patients who have undergone gastrectomy or gastric bypass procedures (Kim GS, Kim CH, Park JY et al. *Metabolism* 1996; 45:1443–6). These patients are at higher risk for vitamin B12 deficiency, due to the lack of intrinsic factor and acidic conditions produced by the stomach, both of which are needed for absorption of vitamin B12.
- Vitamin B12 deficiency may result in suppressed activity of osteoblasts and decreased alkaline phosphatase, which may have an impact on BMD (Kim GS, Kim CH, Park JY et al. *Metabolism* 1996;45:1443–6).
- High homocysteine levels (a consequence of low B12 status) have also been associated with reduced BMD, possibly because they interfere with collagen cross-linking (Dhonushe-Rutten RA, Pluijijm SM, de Groot LC et al. *J Bone Miner Res* 2005;20:921–9; McLean RR, Jacques PF, Selhub J et al. *N Engl J Med* 2004;350:2042–9). Since vitamin B6 and folate are also involved in homocysteine metabolism, one of these other B vitamins may affect BMD more than vitamin B12.

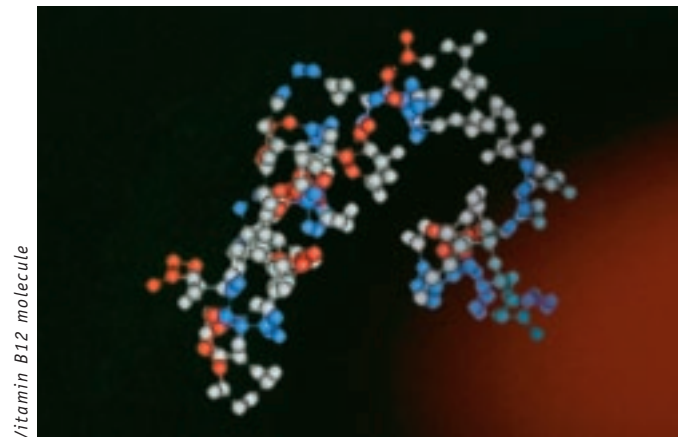
Despite some evidence that vitamin B12 may be important for maintaining BMD, no randomized controlled trials have been carried out.

Jennifer Dunne, RD, completed her undergraduate degree in nutrition at the University of Manitoba and her dietetic internship at Vancouver General Hospital. She has worked in a variety of clinical areas and is currently pursuing her Master's degree in the area of nutrition and bone health.

Dr. Suzanne Morin is an Internist in the Bone Metabolism Centre at the McGill University Health Center (MUHC) and Associate Professor in the Department of Medicine at McGill University.

Dr. Tim Murray is an endocrinologist at St. Michael's Hospital, and Professor of Medicine in the Division of Endocrinology and Medicine at the University of Toronto.

The dietary recommendation (RDA) for vitamin B12 for adults is 2.4 µg per day. For most people who eat a wide variety of foods from all food groups, a balanced diet should provide enough of this vitamin. But since vitamin B12 is found primarily in foods of animal origin, those who consume few or no animal products may be at risk. One study found that poor vitamin B12 status was associated with low BMD in adolescents who had been fed a strict macrobiotic diet during their first years of life (Dhonushe-Rutten RA, van Dusseldorp M, Schneede J. *Eur J Nutr* 2005;44: 341–7). A supplement would be advisable in people consuming a vegetarian diet, or even those who just avoid meat, to ensure adequate vitamin B12 status. There may also be concern in older people, due to the 10%–30% prevalence of malabsorption of naturally occurring vitamin B12 caused by low stomach acid in this population.



Vitamin B12 molecule

All people over 50 years of age should meet their vitamin B12 requirement by consuming foods fortified with vitamin B12 or taking a vitamin B12-containing supplement, such as a multivitamin (unlike in the United States, few foods in Canada are fortified with vitamin B12).

q.

Is there evidence that beta blockers can reduce the risk of bone fractures?

Dr. Suzanne Morin answers: Studies have shown that the sympathetic nervous system can modulate bone metabolism. In general, increased sympathetic nervous activity is thought to increase bone resorption and decrease bone formation. Osteoblasts have been found to have beta2-adrenergic receptors, and administration of beta2-agonists in mice resulted in bone loss. When the mice were given a beta blocker, propranolol, the opposite effect — an increase in bone mass — was documented.

Beta blockers have been used widely for the treatment of cardiovascular diseases, and might provide improvement in bone strength and reduction in the risk of fractures related to osteoporosis. Pasco and colleagues evaluated the association of beta blocker use and fractures in a case-control study in women 50 years and older enrolled in the Australian Geelong Osteoporosis Study (Pasco JA, Henry MJ, Sanders KM et al. *J Bone Miner Res* 2004;19:19–24). They reported a decrease in the rate of any fractures in women on beta-blockers (OR 0.68; 95% CI, 0.49–0.96) even after adjusting for weight, age, medications, diet and lifestyle factors. Further, BMD was found to be higher in the treated group. Another case-control study derived from the UK-based General Practice Research Database also found a positive association between beta blocker use and risk of fracture (OR 0.77; 95% CI, 0.72–0.83) after adjustment for age, sex, body mass index, comorbidities and other medication use (Schleinger RG, Kraenzlin ME, Jick SS et al. *JAMA* 2004;292:1326–32).

A recent report by Reid et al, however, on the prospective cohort of women enrolled in the Study of Osteoporotic Fractures (SOF), showed no clear association between beta blocker use, BMD and fracture risk (Reid IR, Gamble GD, Grey AB et al. *J Bone Miner Res* 2005;20:613–8). The SOF followed a total of 8,412 women prospectively for a mean duration of seven years; 1,099 women who were recorded as users had higher weight, more thiazide, statin and estrogen use, and less glucocorticoid use compared to nonusers. They also smoked less. Over the follow-up period, 2,167 total fractures occurred (of these, 431 were at the wrist and 585 at the hip). Among participants taking beta blockers, the hazard ratio for any fracture was 0.92 (95% CI 0.81–1.05) and for hip fracture, 0.76 (95% CI 0.58–0.99). Adjustments for weight and other confounders did not alter these results. Further, there was no association between beta blockers and BMD after adjustment for weight.

In summary, in the absence of a randomized controlled study, the observational data available to date do not support a beneficial effect of beta blockers on BMD, nor on fracture risk. Additional investigations are needed to reconcile the results of basic research and the apparent lack of clinical effect.

q.

What is the incidence of osteoporosis in patients with celiac disease (CD)? Should these patients be screened for osteoporosis, and what is the best way to manage the risk of bone loss?

Dr. Tim Murray explains: Osteoporosis is often present without any apparent symptoms long before fractures occur. The question of screening is extremely important,

Following a gluten-free diet is the best way to manage the risk of bone loss in patients with celiac disease

as fractures can often be prevented with timely and appropriate treatment.

The incidence of osteoporosis in people with CD is significantly higher than in the general population. While the prevalence in adult CD patients has not been studied in large numbers, available figures suggest that it is between 26% and 34%, compared to findings of 15% for women and 7% for men over the age of 50 in general Canadian population studies. More CD patients will have low BMD of lesser severity. The incidence of fractures is about twice as high in celiac patients as in the general adult population, and even greater in those who are not on a gluten-free diet (GFD). Osteoporosis is also more common in patients who have classic CD bowel symptoms or anemia.

The best way to manage the risk of bone loss is to maintain a GFD. In children or adolescents, a GFD will restore bone mass and bone growth to normal within a year, so screening for osteoporosis is not necessary in these age groups. Virtually all adult patients newly diagnosed with CD will have a bone mass that is at least somewhat below normal. In these patients, we generally recommend measuring BMD to establish the diagnosis of osteoporosis, or simply to indicate a low bone mass that is not yet in the osteoporosis range. A history of previous fractures makes the diagnosis of osteoporosis more likely.

Besides following a GFD, additional measures include a diet rich in calcium and vitamin D supplements. Unless there have been fractures in the past, or BMD is extremely low, no other management may be necessary in premenopausal women or in men under 50 years of age. After that time, the incidence of osteoporosis increases in the general population. Similarly, osteoporosis may develop in CD patients in an age-related manner, for other reasons in addition to the CD. The chances of developing osteoporosis remain higher than in those without CD, unless they are following a GFD.

In some CD patients, specific drug treatment may also be necessary. Physicians assess the diagnosis of osteoporosis and the need for drug therapy based on a combination of factors, some of which include BMD, family history of fracture, age and lifestyle. Again, the most essential strategy if bone mass is low is to follow a GFD — for most adult CD patients, this alone will result in significant improvement. ●



Osteoporosis Canada

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OSTEOPOROSIS CANADA/ISCD SYMPOSIUM

Saturday, April 22, 2006

Holiday Inn Select Halifax Centre, Halifax, NS

8:00–10:00 am (registration and breakfast 7:30–8:00 am)

Organized by Osteoporosis Canada in conjunction with the International Society of Clinical Densitometry certification course (www.iscd.org). The symposium is open to those attending the ISCD course and community family physicians. Registration is required.

“Osteoporosis: What’s now? What’s new?”

Osteoporosis Canada recommendations for BMD reporting: presentation and interpretation of new recommendations published in the Canadian Association of Radiology Journal

Osteoporosis in men: risk factors, clinical assessment and management

Treatments: current and new

Osteoporosis Canada recommendations for optimal reporting of vertebral fractures detected on lung/spine x-rays

Please RSVP by April 15, 2006 to:

Ellen Hunter, 1-800-463-6842

ehunter@osteoporosis.ca

IOF WORLD CONGRESS ON OSTEOPOROSIS

June 2–6 2006

Toronto, ON

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For information and to register:

IOF Congress Secretariat

73, cours Albert Thomas, 69447 Lyon cedex 03, France

Fax: +33 4 72 36 90 52

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www.osteofound.org/wco/2006

about Osteoporosis Canada

Osteoporosis Canada is a national, not-for-profit organization dedicated to educating, empowering and supporting individuals and communities in the prevention and treatment of osteoporosis.

The organization, guided by its Scientific Advisory Council (SAC) made up of osteoporosis experts from across the country, works with healthcare professionals to make the latest prevention, diagnostic and treatment options available to Canadians.

www.osteoporosis.ca