

OSTEOPOROSIS PERSPECTIVES

VOLUME 1

NUMBER 2

Osteoporosis and bone quality

The diagnosis of osteoporosis requires an assessment of risk factors, the documentation of fractures, an evaluation of potential secondary causes of bone loss and, most importantly, measurement of bone mineral density (BMD). Studies have demonstrated a direct relationship between bone density and bone strength¹ and reductions in BMD in postmenopausal women are associated with an increased risk of fracture.²

However, there is a growing awareness that a reduction in bone mineral density is not the sole pathology underlying osteoporosis, nor do increases in BMD completely explain successful therapy. Patients with similar BMDs may have significantly different fracture risks; and agents with differing effects on BMD may produce similar reductions in fracture risk.³ The missing factor appears to be bone *quality*. Legrand et al examined the relationship between the quality of trabecular bone and vertebral crush fractures in 44 male patients with osteoporosis.⁴ There were no significant differences in BMD in patients with or without fractures. However, patients with at least one vertebral fracture had significant alterations in trabecular bone architecture compared with those who were fracture-free. The study suggests that altered trabecular bone architecture is a major determinant of osteoporotic fracture risk in men.



As the studies discussed below suggest, a universally accepted definition of bone quality does not exist. Several factors may be involved; the most important is probably the microarchitecture of bone. In high quality bone, the trabeculae are greater in number, thicker, more platelike, and better connected.³ To these characteristics,



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Edited by
Margery Gass, MD
Professor of Clinical Obstetrics and Gynecology
University of Cincinnati College of Medicine
Director, University Hospital Menopause and
Osteoporosis Center, Cincinnati, OH
President, North American Menopause
Society (NAMS)

Schnitzler adds higher mineralization and less fatigue damage (which is influenced by turnover rate).⁵ Improved secondary mineralization, changes in cortical porosity, and the health of osteocytes may also play roles in the quality of bone.³ Bone quality, as well as quantity, declines with age. The trabecular network becomes progressively disconnected and weaker. Old osteocytes die, leading to hypermineralization and brittleness. Bone collagen becomes unstable and unremodeled bone acquires accumulated fatigue damage.⁵

Effects of osteoporosis therapies on bone quality

Many osteoporosis therapies have been found to affect bone quality as well as its mineral density. Turner notes that anabolic therapies, such as parathyroid hormone (PTH; also known as teriparatide), increase bone turnover and porosity, which can offset some of the positive effects on bone strength. Antiresorptive therapies reduce bone turnover, causing increased bone mineralization, which can increase brittleness.⁶ However, recent studies, both animal and human, suggest that the preservation or improvement of bone microarchitecture accounts for an important part of the benefits of several current osteoporosis medications. In a study by Borah et al, the effects of risedronate on bone mass and architecture were evaluated in ovariectomized minipigs. The animals were treated daily for 18 months with either vehicle or risedronate at doses of 0.5 mg/kg/day or 2.5 mg/kg/day. Bone architecture was measured by 3-D microcomputed tomography. Bone volume was higher in both treated groups ($p < 0.05$), but bone architecture changes were more significant at the 2.5 mg/kg/day dose. At the higher dose of risedronate, trabecular thickness, trabecular number, and connectivity were higher and trabecular separation was lower compared with animals treated with vehicle ($p < 0.05$). Both normalized maximum load (an index of strength) and normalized stiffness of vertebral cores were higher in the 2.5 mg/kg/day group compared with the vehicle group ($p < 0.05$). Vertebral bone volume alone accounted for 76% of the variability in bone strength, while the combination of bone volume and architectural variables accounted for more than 90% of bone strength. The investigators concluded that risedronate preserved trabecular architecture and that bone strength is tightly coupled to both bone mass and architecture.

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PROGRAM FACULTY

Margery Gass, MD
Professor of Clinical Obstetrics
and Gynecology
University of Cincinnati College
of Medicine
Director, University Hospital Menopause
and Osteoporosis Center,
Cincinnati, OH
President, North American Menopause
Society (NAMS)



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STATEMENT OF EDUCATIONAL NEED

This activity is designed to respond to
the needs of obstetricians/gynecologists
and other physicians who care for women
at risk for osteoporosis or with established
osteoporosis.

EDUCATIONAL OBJECTIVES

After reading this newsletter and
completing the post-test, the participant
should be able to:

- Describe the factors that determine
bone quality and the relationship of
bone quality to osteoporosis.
- Describe the effects of the discontinu-
ation of estrogen on bone density and
bone markers.
- Compare the relative effects of
antiresorptive agents on spine and
hip BMD.
- Appreciate the importance of
adopting a more aggressive approach
to the diagnosis of osteoporosis.

STATEMENT OF EDUCATIONAL METHOD

The educational information is presented
in an 8-page newsletter.

STATEMENT OF EVALUATION INSTRUMENT

A 10-question, multiple-choice post-test
is used as the evaluation instrument. An
activity evaluation questionnaire will be
completed by each participant.

STATEMENT OF INTENDED, OR TARGET, AUDIENCE

This activity is intended for, but not
limited to: obstetricians/gynecologists
and other physicians who care for
patients with osteoporosis or who are
at risk for osteoporosis.

INSTRUCTIONS

To earn 1 hour of category 1 credit, read
the material in this newsletter carefully.
Complete the activity evaluation and
answer the post-test questions on the
accompanying questionnaire. Send the
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ANSWER KEY TO CME POST-TEST

1. B 2. E 3. A 4. A 5. A
6. A 7. D 8. A 9. E 10. B

In a three-year trial, biochemical and histological studies assessed bone quality and turnover in women randomized to placebo or alendronate 5 or 10 mg/day for three years or 20 mg/day for two years, followed by 5 mg/day for one year.⁷ All patients also received 500 mg/day of calcium carbonate. Transiliac bone biopsies were obtained from 231 patients from Phase III alendronate studies at the end of either 24 or 36 months of continuous treatment. In patients receiving active treatment, decreased bone resorption was followed by decreases in bone formation. A steady state of bone turnover was achieved after six months of treatment. All 231 biopsy samples were evaluated for the presence or absence of qualitative abnormalities. The investigators found that alendronate did not impair bone mineralization, induce the formation of woven bone, marrow fibrosis, or focal osteomalacia, or have any other adverse effects on bone quality.

In a similar three-year trial, the effects of oral risedronate 5 mg/day on bone quality and remodeling were assessed in 55 women (27 placebo and 28 risedronate).⁸ Transiliac bone biopsies were obtained at baseline and after treatment. The biopsy samples showed no undesirable qualitative changes, such as osteomalacia, peritrabecular fibrosis, or woven bone, associated with treatment.

The effects of alendronate on bone quality and turnover were also studied in secondary osteoporosis.⁹ This study included 52 women and 36 men aged 22-75 years who had long-term glucocorticoid exposure. Patients were randomized to receive placebo or oral alendronate 2.5, 5, or 10 mg/day for one year. Transiliac bone biopsies were then obtained for quantitative and qualitative analysis of bone. In addition to the anticipated decrease in bone turnover, the investigators found that alendronate treatment was not associated with any qualitative abnormalities. There were no differences between the placebo and alendronate groups in trabecular bone volume or parameters of microarchitecture.

Parathyroid hormone (PTH) is a bone-formation stimulating agent; it not only increases bone mass, but also seems to

restore bone architecture by filling in cavities and cancellous bone. The effects of recombinant parathyroid hormone on bone quality differ with duration of treatment. A study of short-term PTH use (56 days) was conducted in 2-year-old male rats treated with daily injections of 15 nmol/kg PTH or vehicle.¹⁰ Rats treated with PTH showed a substantial increase in the strength of the vertebral body compared with those treated with vehicle. Furthermore, a biomechanical analysis showed that compressive bone strength was enhanced, even after correcting for increased bone mass. This suggests that PTH improved bone quality as well as mass.



Another animal study suggested that long-term treatment with PTH may have deleterious effects on bone quality.¹¹ Young female rats received near-lifetime treatment with recombinant PTH at doses of 5, 30, or 75 µg/kg/day or vehicle controls for up to two years as part of an oncogenicity evaluation. Substantially increased bone mass was observed for all treatment groups. However, PTH stimulated osteoblasts and skeletal growth throughout the treatment duration, resulting in abnormal bone architecture and undesirable biomechanical properties. In particular, there was an absence of distinction between trabecular and cortical bone, and the femoral midshaft showed reduced toughness and increased brittleness. The investigators concluded that PTH skeletal effects are a complex function of dose and duration and that, in rats, short-term treatment (six months or less) is more advantageous than near-lifetime treatment.

Dempster et al examined the effect of daily treatment with recombinant PTH on bone microarchitecture and turn-over in patients with osteoporosis.¹² They obtained paired iliac crest bone biopsy specimens from patients with osteoporosis before and after treatment with daily injections of 400 U of recombinant PTH. The first group of eight men was treated with PTH for 18 months. The second group of eight postmenopausal women was treated with PTH for 36 months. The women were maintained on hormone replacement therapy for the duration of the trial. Results showed that can-

cellous bone area was maintained in both groups, while cortical width was maintained in men and significantly increased in women. There was no increase in cortical porosity. There was also an increase in trabecular connectivity density in the majority of patients. The investigators concluded that daily PTH has an anabolic effect on cortical bone in patients with osteoporosis and also improves cancellous bone microarchitecture.

Arzoxifene, a new selective estrogen-receptor modulator (SERM), has also been shown to maintain bone quality as well as BMD. The effects of arzoxifene 0.1 mg/day and 0.5 mg/day were examined in four-month-old ovariectomized rats and compared with controls.¹³ Both doses of arzoxifene prevented ovariectomy-induced declines in BMD. They also maintained bone formation indices and preserved trabecular number above controls. Compression testing and three-point bending testing of the femoral shaft confirmed that bone strength and toughness were higher for treated animals.

Fluoride may also have beneficial effects on bone quality.¹⁴ When prescribed for the prevention of osteoporosis, fluoride modifies the microscopic structure and biomechanical properties of bone. It stimulates bone formation, leading to trabecular hypertrophy and possibly improving interconnections within the trabecular network. However, when the concentration of fluoride in bone becomes excessive, it can lead to mineralization defects; these weaken the bone despite an increase in mass. Thus the benefits of fluoride in preventing vertebral fractures are probably the result of a balance between increases in trabecular bone mass and alterations in bone mineralization.¹⁴

The future: practical clinical techniques for measuring bone quality

Once the characteristics that determine bone quality are established, it will be desirable to develop scales for measuring and quantifying bone quality. These may ultimately prove useful for diagnosis, selecting appropriate osteoporosis therapy, and assessing the results of treatment.^{3,15-17}

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The next step will be the development of practical, noninvasive techniques for assessing bone quality. Although there are several effective techniques for measuring the quality of resected bone, such as multiple spin echos,¹⁷ noninvasive techniques for assessing bone microarchitecture have not yet been perfected.³ In a study published in 1999, Matsubara et al experimented with such a technique, using a morphological filter and pipeline analysis applied to computed radiography (CR).¹⁸ On the basis of trabecular thickness, they divided observed trabecular patterns into eight subsets. They subsequently developed criteria relating the percentage of thicker trabeculae to the strength of the bone. They were then able to correlate an abstracted percentage of thick trabeculae observed by CR to bone strength. By contrast, BMD alone correlated poorly to bone strength.

In summary, our conception of osteoporosis as a disease of low bone mass has moved toward a broader understanding that bone strength is based on both bone quantity and quality.¹⁹ A 1991 consensus conference developed a new definition of osteoporosis as a disease characterized by “low bone mass and microarchitectural deterioration.”²⁰ Current techniques for assessing microarchitectural deterioration are limited by their invasiveness. In the future, the diagnosis of osteoporosis will probably involve more accurate assessments of bone strength using noninvasive methods to measure both bone mineral density and its architectural integrity.¹⁹

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Preliminary EFFECT results: once-weekly alendronate superior to raloxifene for spine and hip BMD

Preliminary results of a year-long study of 456 women have shown that alendronate 70 mg once-weekly produces significantly greater increases in bone mineral density (BMD) of the lumbar spine and total hip than raloxifene 60 mg once-daily.¹ The study results were presented at the 51st Annual Meeting of the American College of Obstetricians and Gynecologists (ACOG).

The Efficacy of Fosamax® vs. Evista® Comparison Trial (EFFECT) enrolled 456 women at 52 centers and randomized them to alendronate (n=223) or raloxifene (n=233). Patients also received calcium 500 mg and vitamin D 400 IU daily. The mean age of enrolled patients was 64 years (range: 37-89 years). All patients had osteoporosis, as defined by a T-score of 2.0 SD or greater below the mean for young adults. The mean baseline lumbar spine T-score was -2.50. A history of fracture was reported by 53%.

Bone mineral density was measured at baseline, at six months, and at 12 months. For the primary endpoint of percent change in BMD at the lumbar spine at one year, there was more than a two-fold increase in patients receiving alendronate compared with those receiving raloxifene (4.4% vs. 1.9%, respectively; $p < 0.001$). Similarly, total hip BMD increased 2.0% for patients on alendronate and 1.0% for patients on raloxifene at one year ($p < 0.001$). In addition, the BMD at the hip trochanter increased 3.2% in the alendronate arm and 1.8% in the raloxifene arm ($p < 0.001$). The response rate, defined as the number of patients who maintained or increased BMD was 94% for alendronate and 75% for raloxifene. Significantly greater increases in BMD at the lumbar spine and total hip were also seen in alendronate patients at the six-month data point ($p < 0.001$ for the spine; $p < 0.013$ for the hip).

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Osteoporosis continues to be underdiagnosed — even in patients with fractures

Despite advances in diagnostic technology, osteoporosis remains an underdiagnosed disease. In a recent retrospective cohort study of 206 patients (146 female, 60 male) with radiographic findings of vertebral compression fractures, only 38% (46% of women and 19% of men) had already been diagnosed with osteoporosis.¹ Furthermore, only 32% (39% of women and 14% of men) received prescription medications for osteoporosis. Many of the patients had several risk fractures for osteoporosis, including 68% of the women and 48% of the men, who had multiple compression fractures of the vertebrae.

Women younger than 50 (adjusted odds ratio = 0.09; 95% CI = 0.01-0.71) and 90 or older (AOR = 0.27; 95% CI = 0.08-0.98) were less likely to have been diagnosed with osteoporosis. Women with a prior hip or radial fracture (AOR = 3.65; 95% CI = 1.28-10.38) or back pain (AOR = 2.84; 95% CI = 1.38-5.85) were more likely to have been diagnosed with osteoporosis.

The authors concluded that, in the primary care setting, physicians frequently did not diagnose osteoporosis in patients with vertebral fractures, thereby missing an opportunity to prevent future fractures in patients at high risk. They called for targeted efforts to improve diagnosis as an important step in enhancing patient care. The National Osteoporosis Foundation (NOF) currently recommends that all postmenopausal women with fractures be evaluated for osteoporosis, including BMD measurements. The NOF also recommends BMD testing for all women aged 65 and for postmenopausal women younger than 65 with one or more additional risk factors.

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NOF recommends more aggressive approach to diagnosis and treatment of low BMD



The National Osteoporosis Foundation (NOF) recently updated its *Physician's Pocket Guide to Prevention and Treatment of Osteoporosis* to reflect new treatment options and to encourage more extensive diagnosis and treatment of patients at risk for fractures.¹ Perhaps the most significant change is the recommendation to initiate therapy in postmenopausal women with BMD T-scores below -2 in the absence of risk factors. (The previous recommendation was to treat if T-scores were below -2.5.) The updated *Guide* also recommends treatment if T-scores are below -1.5 if one or more risk factors are present (*Table 1*).

The more aggressive approach to fracture prevention is, in part, a response to the long-awaited results of the National Osteoporosis Risk Assessment (NORA) trial, the largest U.S. study of osteoporosis conducted to date.² NORA was a longitudinal observational study conducted from September 1997 to March 1999, with approximately 12 months of subsequent follow-up. The study enrolled 200,160 postmenopausal women aged 50 or older, who had not been previously diagnosed with osteoporosis. The principal outcome measures were baseline BMD T-scores obtained through peripheral bone densitometry or ultrasonography of heel, finger, or forearm, and clinical fracture rates at the 12-month follow-up.

The prevalence of osteopenia and osteoporosis among women enrolled in the study was surprisingly high. Based on the criteria of the World Health Organization, 39.6% of the enrolled patients had osteopenia (T-scores of -1 to -2.49) and 7.2% had osteoporosis (T-scores ≤ -2.5). Follow-up data were available from 163,979 patients. Among patients with osteoporosis, there was a four-fold increase in fracture rate (95% CI; 3.59-4.53)

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Table 1. Risk factors for osteoporotic fracture¹

- Personal history of fracture as an adult
- History of fragility fracture in a first degree relative
- Low body weight (< about 127 pounds)
- Current smoking
- Use of corticosteroid therapy for more than 3 months
- Impaired vision
- Estrogen deficiency at an early age (< 45 yrs)
- Dementia
- Poor health/frailty
- Recent falls
- Low calcium intake (lifelong)
- Low physical activity
- Alcohol intake in excess of 2 drinks per day

compared with that seen in patients with normal BMDs. Among patients with osteopenia, there was a 1.8-fold increase in fracture rate (95% CI; 1.49-2.18).³

The NORA results suggest that much more can and should be done to identify and treat patients at risk for osteoporotic fractures. Almost half of the patients enrolled in this study had previously undetected low BMDs. Perhaps most alarming is the observation that patients with T-scores of -1 to -2.49, who would not have received treatment under previous NOF guidelines, had a fracture risk almost double that of patients with normal BMDs. In a commentary on NORA, Chesnut notes that "...NORA confirms what many clinicians and osteoporosis researchers have long suspected, i.e., that a significant number of postmenopausal women in pri-

mary care practices have clinically significant low BMD and that such women have an increased risk of incident fracture within one year."⁴ Given the personal, economic, and social impact of osteoporotic fractures, a more aggressive approach to diagnosis and treatment is clearly justified.

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ABSTRACT

The following abstract was presented at the ACOG 51st Annual Clinical Meeting, April 26-30, 2003, New Orleans, Louisiana.

Osteoporosis counseling study: a randomized clinical trial

Mark A. Binstock, MD, MPH
Margaret F. Griffin, MSN, CNP

OBJECTIVE

Compliance with osteoporosis medications is low, similar to therapy for other chronic disorders. The purpose was to investigate the impact of an intensive counseling session by a specially trained nurse on therapy compliance, satisfaction, and therapy costs among patients undergoing bone density testing (DXA).

METHODS

74 post-DXA patients who met treatment thresholds (National Osteoporosis Foundation and/or high risk for hip fracture) were offered participation. They were randomized 3:1 between the intensive counseling (IC) (57 patients) and usual care (UC) (17 patients). Patients in both groups received individualized reports summarizing their results and treatment recommendations along with a 24-page pamphlet. Usual Care patients were instructed to contact their ordering provider to review results and discuss therapy. Intensively Counseled patients received concurrent counseling by a nurse including: indications, therapy advised, risks and/or side effects, costs, enrollment in manufacturer's drug assistance program

as needed, and issuance of a prescription. All patients were surveyed at 1 month. Pharmacy costs and patient drug co-payments were assessed.

RESULTS

IC patients had higher rates of bone protective drug use (79% versus 65%), satisfaction with care experience (8.4% versus 8.1%), and drug assistance program enrollment (54% versus 0%), and lower out-of-pocket drug costs per patient (\$64 versus \$73) and health plan drug acquisition costs per patient (\$191 versus \$215) than UC patients.

CONCLUSION

Structured counseling after DXA by a specially trained nurse leads to improved medication compliance and patient satisfaction while reducing drug copayments and health plan acquisition costs.

CASE STUDY 1

A 65-year old woman with bone loss and celiac disease

Submitted by Marjorie M. Luckey, MD
 Director, Saint Barnabas Osteoporosis and Metabolic Center, Livingston, NJ;
 Associate Clinical Professor,
 Mount Sinai School of Medicine,
 New York, NY

PATIENT PROFILE

Mary R. is a 65-year-old healthy Caucasian woman (height 5' 2", weight 125 lbs), who recently retired as a secretary. She has had six pregnancies with normal outcomes. Menopause began at age 50; she has never taken estrogen. Her history includes long-standing symptoms of irritable bowel syndrome. She has never smoked and her alcohol intake is minimal. She takes a multi-vitamin, 600 mg calcium carbonate, and 200 IU vitamin D daily. She also drinks one glass of milk and one glass of calcium-fortified orange juice daily. Her bone mineral density (BMD) results are listed in the table below.

Table 1. BMD Results

	T-scores	Z-scores
Spine (L1-4)	-3.8	-2.5
Total hip	-3.0	-2.0

Lab results were normal for serum calcium, phosphorus, alkaline phosphatase, CBC, and differential. Her 25 OH vitamin D was 28 ng/mL and 24-hour urine calcium was 30 mg/24 hr. Tests for antigliadin antibodies and transglutaminase antibody were strongly positive. A small-bowel biopsy confirmed the presence of celiac disease.

DISCUSSION

This patient's low Z-score suggests that something other than (or in addition to) postmenopausal bone loss is occurring.

The patient's long-standing irritable bowel syndrome (IBS) is an important clue: a major proportion of IBS patients have gluten sensitivity. Celiac disease is an inherited disorder caused by intolerance to the gliadin fraction of gluten. Gliadin combines with antibodies, forming an immune complex that damages the intestinal mucosa. The disease has a major impact on calcium absorption. Her 24-hour urine calcium confirmed the presence of calcium malabsorption. Thus, Mary R.'s celiac disease must be addressed before her bone loss can be treated effectively. This case underscores the importance of a laboratory work-up to rule out secondary causes of osteoporosis.

CASE STUDY 2

A 45-year old high school teacher with seizures and asthma

Submitted by Marjorie M. Luckey, MD

PATIENT PROFILE

Susan T. is a 45-year-old African American high school teacher. Her history includes surgical menopause five years previously. She never took estrogen and currently has mild vasomotor symptoms only. She was diagnosed with a seizure disorder at age 20 and is currently well controlled on phenytoin 300 mg daily. She has had asthma since childhood; her current medications include a b.i.d. steroid inhaler and oral glucocorticoids 5-6 times per year for 2-6 weeks for exacerbations. She takes a multi-vitamin daily, Tums® 500 mg b.i.d., and has one dairy serving daily. She has no history of osteoporotic fractures.

Table 2. BMD Results

	T-scores (vs. Caucasian women)	T-scores (vs. African American women)	Z-scores
Spine	-2.0	-2.8	-2.4
Total hip	-1.5	-2.6	-2.0

Her physical exam results showed a height of 5'5" (no loss), a weight of 150 lbs, and a BP of 140/82. Expiratory wheezes were noted. Her lab test results showed the following: sCa: 8.9 (normal: 8.6-10.2), normal CBC, normal cholesterol, 24-hour urine calcium: 50 mg, and 25 OH vitamin D: 8 ng/mL (normal: >20). Her BMD results are listed in the table below.

DISCUSSION

This patient's BMD is much lower than would be expected for her age, which suggests a secondary cause for her bone loss. Although African American women are less prone to develop osteoporosis than Caucasian women, this case underscores the importance of obtaining BMD scores in all ethnic groups. All women should be screened by age 65, while those with risk factors, such as exposure to drugs that may cause bone loss, should be screened earlier. Compared with normal mean BMDs for African American women, Susan's T-score suggests that she is at significant risk for fractures, while her Z-score suggests a secondary cause of bone loss. In fact, she has several risk factors for secondary osteoporosis, including early surgical menopause and chronic exposure to anticonvulsants and corticosteroids. In this case, the primary culprit was a vitamin D deficiency: vitamin D is critical for calcium absorption. In addition to reducing calcium absorption, vitamin D deficiency has adverse neuromuscular effects that significantly increase the risk of falls and fractures. It has recently been recognized that 15-20% of the Caucasian population has vitamin D deficiency, and the incidence is even higher in the African American population. One cause of vitamin D deficiency is anticonvulsant

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EFFECT results *continued*

Risa Kagan, MD, co-medical director of FORE-Foundation for Osteoporosis Research, noted that there were no clinically apparent vertebral or hip fractures in either arm, but there were a variety of fractures of the wrist, shoulder, or toes; these data will be presented at a later date. The discontinuation rate was 11.2% in the alendronate arm and 1.3% in the raloxifene arm.

REFERENCES

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Case Study 2 *continued*

therapy; many anticonvulsants, including phenytoin, interfere with the metabolism of vitamin D. In general, people under age 70 should receive 400 units of vitamin D daily; for those 70 and older, the recommended dose is 600-800 units, while patients with osteoporosis should have a daily vitamin D intake of 800-1000 units.

In summary, this case is a timely reminder that not all low bone density is simple osteoporosis. Several potential causes of secondary osteoporosis needed to be addressed before she could be treated successfully for bone loss.

POST-TEST

<p>1. Successful osteoporosis therapy can be entirely accounted for by increases in bone mineral density.</p> <p><input type="checkbox"/> A. True <input type="checkbox"/> B. False</p> <p>2. Which of the following is (are) consequences of aging on bone?</p> <p><input type="checkbox"/> A. Accumulated fatigue damage <input type="checkbox"/> B. Collagen becomes unstable <input type="checkbox"/> C. Hypermineralization <input type="checkbox"/> D. Disconnection of the trabecular network <input type="checkbox"/> E. All of the above <input type="checkbox"/> F. Only A, B, and D above</p> <p>3. Long-term use of PTH may have undesirable effects on bone.</p> <p><input type="checkbox"/> A. True <input type="checkbox"/> B. False</p> <p>4. One of the drawbacks of current approaches for assessing bone quality is the absence of noninvasive techniques.</p> <p><input type="checkbox"/> A. True <input type="checkbox"/> B. False</p>	<p>5. In the EFFECT trial, alendronate was more than twice as effective as raloxifene in increasing lumbar spine BMD at 12 months.</p> <p><input type="checkbox"/> A. True <input type="checkbox"/> B. False</p> <p>6. In the retrospective cohort study by Neuner et al, what percentage of women with radiographic evidence of vertebral compression fractures had previously been diagnosed with osteoporosis?</p> <p><input type="checkbox"/> A. 46% <input type="checkbox"/> C. 86% <input type="checkbox"/> B. 74%</p> <p>7. What is (are) the current recommendations of the National Osteoporosis Foundation for initiating osteoporosis therapy in postmenopausal women?</p> <p><input type="checkbox"/> A. A T-score below -2 in the absence of risk factors <input type="checkbox"/> B. A T-score below -2.5 in the absence of risk factors <input type="checkbox"/> C. A T-score below -1.5 if one or more risk factors are present <input type="checkbox"/> D. A and C above</p>	<p>8. The NORA study showed that patients with T-scores of -1 to -2.49 had a fracture risk almost double that of patients with normal BMDs.</p> <p><input type="checkbox"/> A. True <input type="checkbox"/> B. False</p> <p>9. Which of the following is a potential secondary cause of osteoporosis?</p> <p><input type="checkbox"/> A. Anticonvulsant drugs <input type="checkbox"/> B. Early surgical menopause <input type="checkbox"/> C. Celiac disease <input type="checkbox"/> D. Chronic corticosteroid use <input type="checkbox"/> E. All of the above <input type="checkbox"/> F. Only A, B, and C above</p> <p>10. Structured counseling DXA by a specially trained nurse has not been shown to improve outcomes after DXA.</p> <p><input type="checkbox"/> A. True <input type="checkbox"/> B. False</p>
<p>Seven correct answers are required for credit</p>		

ACTIVITY EVALUATION

Activity Evaluation

1. As a result of the information contained in this activity, will you make any changes in your practice? Yes _____ No _____
If yes, what changes? _____

2. In your opinion, how could this activity be improved? (e.g., change format, more details, fewer details, discuss other topics, change length) _____

3. Please rate the educational value/clinical relevance of this activity.
____ Excellent/outstanding ____ Very good ____ Good/above average
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5. Was the material presented objectively and did it avoid commercial bias?
Yes _____ No _____ Comments: _____

6. Suggestions for future topics: _____

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POST-TEST ANSWERS

O S T E O P O R O S I S P E R S P E C T I V E S

Volume 1  Number 2

Activity Number: END IN 07 101 03

Expiration date: June 2005

To earn one (1) hour of category 1 CME credit after reading this newsletter, please mail the completed post-test answers, activity evaluation, and personal information questionnaire in the enclosed envelope.

Post-Test Answers

(Circle the appropriate letter for each question.)

1. A B 2. A B C D E F 3. A B 4. A B 5. A B 6. A B C
7. A B C D 8. A B 9. A B C D E F 10. A B

