Atraumatic Bilateral Femur Fracture in
Long-term Bisphosphonate Use

To the Editor:

We read with interest the article “Atraumatic Bilateral Femur Fracture in Long-term Bisphosphonate Use” (http://www.orthosupersite.com/view.aspx?rid=41933) by Goddard et al in the August 2009 issue of ORTHOPEDICS.

An increasing number of reports suggest that bisphosphonates may have deleterious effects on bone metabolism, causing stress fractures of femoral diaphysis.1,2 Most of the published case reports, including that of Goddard et al, contain no information on bone metabolism, although some have included indirect measurements of bone metabolism. As far as we are aware, after the original report by Odvina et al,1 there have been no case reports of bilateral femoral fractures including histomorphometric evidence of low bone turnover.

We treated an 80-year-old woman suffering from hypertension and glaucoma. She began treatment with alendronate (70 mg/week) 5 years ago, after rib fractures and an osteopenic finding on dual-energy x-ray absorptiometry (DEXA) scan. Two years later, while picking berries, she sustained a low-energy left femoral diaphyseal fracture (Figure A). The fracture was treated with intramedullary nailing, and alendronate was continued. Three years later, she reported thigh pain on the left side. Cortical thickening was noted on radiographs, and osteoid osteoma was suspected. The patient experienced abrupt, intense pain and was unable to walk. On radiographs, a similar fracture to the contralateral side was noted (Figure B). The fracture was nailed, iliac crest biopsy (without labeling) and tetracycline-labeled bone biopsy were performed, and alendronate was discontinued. During follow-up, both fractures healed well. Both iliac crest biopsies displayed low osteoid formation and cellular indices. There was low tetracycline uptake and no double-labels in the second biopsy. These findings suggest that she had severely suppressed bone turnover.

Concerns have been raised about atypical femoral shaft fractures associated with alendronate use, but reports have included no data about bone metabolism. It has also been suggested that only long (at least 6-7 years) bisphosphonate therapy may cause atypical fractures of the femoral diaphysis. Our case demonstrates that even a shorter duration of treatment may suppress bone metabolism sufficiently to cause stress fractures.

Our patient initially had osteopenia, not osteoporosis, in her DEXA scan. Bisphosphonates are recommended for glucocorticoid-treated patients with osteopenia.3 Although our patient had not been treated with glucocorticoids, we suggest that bisphosphonate treatment should be used with caution in nonosteoporotic patients. The risk of atypical fractures could be increased if patients with nonosteoporotic bone mineral density and an initially low bone turnover are treated with bisphosphonates.

Lastly, Goddard et al addressed the need for additional pathophysiology studies on this subject. Specifically, we suggest that patients acquiring atypical femoral fractures during bisphosphonate therapy should undergo tetracycline-labeled bone biopsy. The etiology behind atypical stress fractures of femurs in our case was severely suppressed bone turnover.

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REFERENCES


Reply:

With more clinicians reporting incidences of atraumatic fractures in patients taking bisphosphonates, we may be better able to ascertain the underlying mechanism of this phenomenon. Identifying appropriate medication holidays, where the beneficial effects of bisphosphonates are maximized while reducing the amount of bone turnover suppression, may be an area of future study.

We agree that more histomorphometric evidence is required in suspected cases. The use of tetracycline-labeled bone biopsies would provide valuable evidence of the degree of active bone remodeling. Given the heightened awareness, physicians should be more likely to include this type of testing when ruling out pathologic causes in patients presenting with low-energy femoral fractures.

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Comparison of Vacuum-assisted Closure to the Antibiotic Bead Pouch for the Treatment of Blast Injury of the Extremity

To the Editor:

We read with interest the article “Comparison of Vacuum-assisted Closure to the Antibiotic Bead Pouch for the Treatment of Blast Injury of the Extremity” (http://www.orthosupersite.com/view.aspx?rid=60134) by Warner et al in the February 2010 issue of ORTHOPEDICS.

The authors report in a small series the economic and logistical advantage of the antibiotic bead pouch in comparison to negative pressure wound therapy for severe extremity injury. The impetus for the use of bone cement bead chains in acute injury came from Dr Klaus Klemm of Frankfurt am Main in 1983. Klemm asked why one would not want high levels of antibiotics in traumatic wounds. The fear that more resistant organisms would be selected was not borne out in Klemm’s own work, which showed that wounds that failed bead therapy generally were infected with sensitive organisms. Warner et al point out that repeat debridement is required to remove nonviable tissue in these blast injuries. The economic advantage of bead chain therapy may be greater than shown in this series since the last work in Klemm’s lab showed that vancomycin does not leach as well as tobramycin and may not be necessary in the formulation of antibiotic bead chains. For many years, traumatologists in the United States have had to resort to “rolling their own” since the device is not approved by the Food and Drug Administration. It is time that these valuable implants, which reliably reduce the incidence of infectious complications in skeletal trauma, can be purchased and implanted in the United States.

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