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Commentary

Osteoporosis and Cortical Bone Remodeling

Bronislaw Slomiany*

Department of Medicine, University of Medicine and Dentistry of New Jersey, New Jersey, USA

DESCRIPTION

Bisphosphonates are anti resorptive medicines used to treat people with osteoporosis who are at risk of fractures. Bisphosphonate therapy lowers fracture risk while also increasing Bone Mineral Density (BMD). Because BMD and bone strength are highly correlated, most studies investigating the effects of bisphosphonate medication have used these metrics as the primary endpoint. Furthermore, higher BMD is a good indicator of lower fracture risk. BMD, on the other hand, does not distinguish between trabecular and cortical bone, and its prognostic power varies by skeletal region. While short-term alendronate treatment has been demonstrated to improve BMD at a variety of skeletal sites, the impact was most strong at trabecular bone sites. As a result, fracture-reduction outcomes differ between vertebral and non-vertebral fractures. Differences in microstructure and metabolic activity between trabecular and cortical bone may be causing this variation between sites. Because of its enormous surface area in close contact with the marrow, trabecular bone is extremely accessible for bisphosphonates, whereas intracortical bone is only accessible through its canal network. As a result, bisphosphonates may have different effects on cortical and trabecular bone.

After treatment with alendronate and risedronate, serum markers of bone resorption C-terminal Telopeptide (CTX) and N-terminal Telopeptide (NTX) decrease, but serum markers of bone formation like P1NP (N-propeptide of type I collagen) and bone alkaline phosphatase decrease, indicating that alendronate reduces bone turnover. Alendronate inhibits bone production, according to histomorphometric examinations of trabecular bone that looked at factors like osteoid/mineralizing surfaces per bone surface and bone activation frequency. On the other hand, there is no good histomorphometric measure of bone resorption, and the ones that have been reported, such as osteoclast/eroded surfaces per bone surface, show no reduction after therapy with alendronate. Because bone production and resorption are generally linked during the bone remodeling process, decrease in bone formation has been interpreted as a consequence of reduction in bone resorption, despite the lack of a strong histomorphometric marker of resorption. Because of the bisphosphonate-induced reduction in bone production, the higher BMD reported in clinical trials is likely due to an enhanced degree of bone matrix mineralization.

In contrast, the bone mass appears to be unaltered. According to a new study on trabecular bone, alendronate treatment resulted in the buildup of halted degraded surfaces without indications of active resorption or surrounding bone growth, indicating a protracted reversal phase. The low number of osteoblastic reversal cells found on arrested eroding surfaces is likely due to an insufficient incorporation of osteoprogenitors, which hampered their transition to formation. When resorption is initiated and pores are extended, cortical pores provide a direct measure of bone loss, according to a histomorphometric analysis relating the pores bone remodeling type and stage directly to cortical porosity (e.g., bone loss). This is in contrast to traditional bone surface-based histomorphometric techniques employed on trabecular bone surfaces, where there are no direct assessments of trabecular bone loss since trabeculae disappear as the bone degrades. As a result, the remodeling activities on the trabeculae can only be linked to the surviving trabecular bone volume, not the actual bone loss.

As the incidence of formative pores decreases in the treated group, alendronate produces an accumulation of degraded holes, indicating a protracted reversal-resorption phase. Bisphosphonates have an effect on both the trabecular and cortical compartments of the brain. Furthermore, the accumulation of eroded holes caused by bisphosphonates, which was regarded as a protracted reversal-resorption phase, was only seen after three years of treatment, implying that treatment duration is important. According to this theory, even lengthier therapy would result in an increased buildup of eroded pores, which would merge and form wider eroded cavities, as seen in a long-term bisphosphonate user's atypical femoral fracture site. More research is needed to confirm the significance of this idea in bisphosphonate-related Atypical Femur Fractures (AFFs).

Correspondence to: Bronislaw Slomiany, Department of Medicine, University of Medicine and Dentistry of New Jersey, New Jersey, USA, E-mail: drslomianyblaw@gmail.com

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