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Magnesium Deficiency Induces Bone Loss in the Rat

Rude R.K.[a] · Kirchen M.E.[a] · Gruber H.E.[b] · Stasky A.A.[b] · Meyer M.H.[b]

[a] University of Southern California and Orthopedic Hospital, Los Angeles, Calif., [b] Carolinas Medical Center, Charlotte, N.C., USA

Abstract

Disorders in which magnesium (Mg) depletion is common have an associated high incidence of osteoporosis.[1] Mg depletion in humans results in hypocalcemia, low serum parathyroid hormone (PTH) and 1,25$(OH)_2$-vitamin D levels, as well as PTH and vitamin D resistance which may serve as mechanisms for the development of osteoporosis. In order to determine if isolated Mg depletion will result in bone loss, we have induced dietary Mg deficiency in the rat. Adult (290 g) female rats were given either a low-Mg diet (2 mg/100 g chow; n = 6) or a normal control Mg diet (63 mg/100 g chow; n = 6). Dietary calcium (Ca) was normal in both groups (592 mg/100 g chow). At 12 weeks, blood was obtained for serum Mg, Ca, PTH, 1,25$(OH)_2$-vitamin D, and osteocalcin determinations. The rats were then euthanized and the femurs obtained for mineral analysis and histomorphometry. Serum Mg in the low-Mg group was less than control (0.4 ± 0.2 vs. 1.9 ± 0.2 mg/dl, $p < 0.001$; mean ± SD) while serum Ca was higher (11.7 ± 0.5 vs. 9.3 ± 0.4 mg/dl, $p < 0.001$). PTH was suppressed in the Mg-deficient group (36 ± 16 vs. 109 ± 30 pg/ml in controls, $p < 0.002$). Serum 1,25$(OH)_2$-vitamin D was also suppressed in the Mg-deficient animals (7.1 ± 4.8 vs. 28.5 ± 8.2 pg/ml in controls, $p < 0.002$). Serum osteocalcin levels were not different (19.8 ± 2.5 ng/ml in Mg-deficient rats vs. 15.3 ± 3.4 ng/ml in controls). While the ash weight of Ca and phosphorus in the femur did not change, the ash weight of Mg fell (low-Mg group 0.55 ± 0.01%, controls 0.65 ± 0.02%, $p < 0.001$). Histomorphometry demonstrated reduction in bone mass; the trabecular bone volume in the femur of the low-Mg group was reduced from control (7.7 ± 0.2 vs. 13.7 ± 1.9%, $p < 0.002$). **A surprising new observation was an increase in osteoclast (OC) bone resorption with Mg depletion.**[2] The number of OC per millimeter bone surface was 16.9 ± 1.3 in the low-Mg group versus 7.8 ± 1.5 in controls ($p < 0.001$). The percentage of bone surface occupied by OC was 38.3 ± 3.7 in the low-Mg group versus 17.7 ± 2.4 in controls ($p < 0.001$). This increased resorption occurred with an inappropriate non-altered bone-forming surface relative to control (% osteoid surface: low-Mg group 2.4 ± 0.7 vs. controls 2.6 ± 0.4; % osteoid volume: low-Mg group 0.25 ± 0.09 vs. controls 0.38 ± 0.06; number of osteoblasts per millimeter bone surface: low-Mg group 0.9 ± 0.3 vs. controls 1.3 ± 0.3). No increase in bone-forming surface or osteoblast number despite an increase in OC-resorbing surface and OC number strongly suggests impaired activation of osteoblasts and an uncoupling of bone formation and bone resorption. **Our data demonstrate that Mg depletion in the rat alters bone and mineral metabolism which results in bone loss.**[3]

Übersetzung (Fettgedrucktes):

[1] Störungen, bei denen häufig ein Magnesiummangel einhergeht, haben ein entsprechend häufiges Vorkommen von Osteoporose.

[2] Eine überraschende Beobachtung war ein Anstieg des Knochenabbaus durch Osteoklasten bei Magnesiummangel.

[2] Unsere Daten zeigen, dass Magnesiummangel bei Ratten den Knochenstoffwechsel ändert und zu Knochenverlust führt.

Osteoklasten sind knochenabbauende Zellen. Osteoblasten sind knochenanbauende Zellen.
Der Knochen wird durch ständigen Ab- und Anbau erneuert. Eine Verschiebung des Gleichgewichts in Richtung Abbau (erhöhte Aktivität der Osteoklasten) führt zu Osteoporose. Die meisten Osteoporosemedikamente (Bisphosphonate) wirken durch Hemmung der Osteoklastenaktivität.