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Impact of GH Administration on Skeletal Endpoints in Adults with Overweight/Obesity

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Background: Obesity is associated with relative growth hormone (GH) deficiency and increased fracture risk. We hypothesized that GH administration would improve bone endpoints in individuals with overweight/obesity.

Methods: 77 adults ages 18-65 years, BMI ≥25 kg/m², and BMD T- or Z-score ≤ -1.0 were randomized in a double-blind protocol to daily subcutaneous GH or placebo for 18 months, targeting IGF-1 in the upper quartile of the age-appropriate normal range. There was a post-treatment observation period from 18-24 months. DXA and high-resolution peripheral quantitative CT were performed at baseline, 18 months and 24 months. Volumetric quantitative CT was performed at baseline and 18 months. Results are reported as mean ±SD or mean (95% confidence interval).

Results: There were no pretreatment differences between the GH (n=39) and placebo groups (n=38), including mean age (48±12y), BMI (33.1±5.7kg/m²) and BMD at any site. Forty-nine subjects (47% female) completed 18 months. P1NP, osteocalcin and CTX increased (p<0.005) and visceral adipose tissue decreased (p=0.04) at 18 months in the GH vs placebo group. Hip and radius aBMD, spine and tibial vBMD, tibial cortical thickness, and radial and tibial failure load decreased at 18 months in the GH vs placebo group (p<0.05). During the post-treatment observation period (18 to 24 months), total radius aBMD and tibia cortical thickness increased in the GH vs placebo group (p<0.05); there was also a trend toward an increase in total hip aBMD in the GH vs placebo group (p=0.06). At 24 months, none of the differences between the GH and placebo groups remained significant. There was a higher incidence of numbness and tingling (33% vs 8%, p=0.01) and joint pain or stiffness (33% vs 5%, p=0.003) in the GH vs placebo group. There were no other differences in adverse events between groups.

Conclusions: We demonstrated that GH administration for 18 months to individuals with overweight/obesity and low BMD decreased some measures of BMD, bone microarchitecture, and bone strength compared with placebo. None of these differences remained significant after 6 months off therapy. A longer duration of treatment, or a longer duration of observation post-treatment, may be necessary to see the expected biphasic decline and then increase in BMD reflecting an expanding remodeling space followed by mineralization that has previously been seen with GH administration in other populations, including individuals with and without GH deficiency. Although future investigation of the effects of GH on bone is required to assess the true long-term impact on skeletal integrity as well as fracture reduction, our study suggests that GH administration for 18 months to adults with overweight/obesity does not improve BMD, bone microarchitecture, or bone strength.

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