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1: [J Endocrinol Invest.](#) 1999 May;22(5):333-9.

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Effects of two years of growth hormone (GH) replacement therapy on bone metabolism and mineral density in childhood and adulthood onset GH deficient patients.

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The aim of the current study was to evaluate bone metabolism and mass before and after 2 years of GH replacement therapy in adults with childhood or adulthood onset GH deficiency. Thirty-six adults with GH deficiency, 18 with childhood onset, 18 with adulthood onset GH deficiency and 28 sex-, age-, height- and weight-matched healthy subjects entered the study. Biochemical indexes of bone turnover such as serum osteocalcin, serum carboxyterminal telopeptide of type-I procollagen, urinary hydroxyproline/creatinine and deoxypyridinoline/creatinine, of soft tissue formation such as aminoterminal propeptide of type-III and bone mineral density were evaluated. Childhood onset GH deficient patients had significantly decreased bone (osteocalcin: 2.5+/-1.3 vs 6.6+/-4.8 mcg/l, p<0.001) and soft tissue formation (aminoterminal propeptide of type III: 273+/-49 vs 454+/-23 U/I, p<0.001) indexes and normal bone resorption indexes (serum carboxyterminal telopeptide of type-I procollagen: 105+/-48 vs 128+/-28 mcg/l p=NS; urinary hydroxyproline/creatinine: 0.19+/-0.16 vs 0.28+/-0.16 mmol/mol, p=NS; urinary deoxypyridinoline/creatinine: 21 +/-10 vs 25+/-8 mcmol/mol, p=NS) compared to healthy subjects. On the contrary, no significant difference in bone turnover indexes between adulthood onset GH deficient patients and healthy subjects was found. Moreover, significantly decreased bone mineral density at any skeletal site and at whole skeleton was found in GH deficient patients compared to healthy subjects (e.g. femoral neck: 0.74+/-0.13 vs 0.97+/-0.11 g/cm², p<0.001). In addition, a significant reduction of bone mineral density was found in childhood compared to adulthood onset GH deficient patients at any skeletal site, except at femoral neck. After 3-6 months of treatment, both groups of patients had a significant increase in bone turnover and in soft tissue formation. In particular, in childhood onset GH deficient patients after 3 months osteocalcin increased from 2.5+/-1.3 to 7.9+/-2.1 mcg/l, p<0.001 aminoterminal propeptide of type-III from 273+/-49 to 359+/-15 U/I p<0.001; serum carboxyterminal telopeptide of type-I procollagen from 105+/-48 to 201+/-45 mcg/l, p<0.001; urinary hydroxyproline/creatinine from 0.19+/-0.16 to 0.81+/-0.17 mmol/mol, p<0.001; urinary deoxypyridinoline/creatinine from 21 +/-10 to 54+/-20 mcmol/mol, p<0.001; while in adulthood onset GH deficient patients after 6 months osteocalcin increased from 4.2+/-3.6 to 6.5+/-1.9 mcg/l, p<0.05; aminoterminal propeptide of type- III from 440+/-41 to 484+/-37 U/I, p<0.05; serum carboxyterminal telopeptide of type-I procollagen from 125+/-40 to

152+/-22 mcg/l, p<0.05; urinary hydroxyproline/creatinine from 0.24+/-0.12 to 0.54+/-0.06 mmol/mol, p<0.001; urinary deoxypyridinoline/creatinine from 23+/-8 to 42+/-5 mmol/mol, p<0.001. No significant difference in bone turnover between pre- and post-treatment period was found after 18-24 months of GH therapy. Conversely, bone mineral density was slightly reduced after 3-6 months of GH therapy, while it was significantly increased after 18-24 months. In fact, femoral neck bone mineral density values significantly rose from 0.74+/-0.13 g/cm² to 0.87+/-0.11 g/cm² (pre-treatment vs 2 years of GH treatment values). In conclusion, patients with childhood or adulthood onset GH deficiency have osteopenia that can be improved by long-term treatment with GH.

PMID: 10401706 [PubMed - indexed for MEDLINE]

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