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HYPOESTROGENISM IN YOUNG WOMEN AND ITS INFLUENCE ON BONE MINERAL DENSITY

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Estradiol is one of the most important hormonal factors responsible for bone health. Genetic factors, adequacy of hormonal functioning, nutrition and physical activity may be the markers of bone status and development in young women. During adolescence, women reach peak bone acquisition and develop a skeletal mass. This process is largely regulated by endocrine factors mainly such as adequate levels of gonadal, adrenal and pituitary hormones. The crucial role played by estradiol and its impact on bones are very multiple. Estradiol induces growth factors' activation, receptor activator of nuclear factor kappa B ligand (RANKL) production inhibition and is mainly referred to antiresorptive activity. Clinical situations leading to hypoestrogenism has been linked to decreased bone mineral density leading to osteopenia and osteoporosis. This status both in fertile and perimenopausal women can increase the risk of pathological fractures. Such conditions as hypothalamic-pituitary insufficiency (functional hypothalamic amenorrhea, anorexia nervosa, Kallmann syndrome, hyperprolactinemia), ovarian failure (gonadal dysgenesis, premature ovarian failure) and iatrogenic treatment (surgery, chemotherapy, radiotherapy) can cause hypoestrogenism. The treatment of osteopenia and osteoporosis caused by hypoestrogenism is very essential and multidirectional. The crucial role of the therapy is the achievement of proper serum estradiol concentration and eliminate the causes of hypoestrogenism.

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