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PRACTICAL APPROACH DIAGNOSTICS OF OSTEOPOROSIS

Abstract: Osteoporosis is a systemic, metabolic skeletal disease characterized by low bone mass and microarhitectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk. It is the most frequent cause of microfractures and bone fracture, deformity and invalidity in old population, especially in postmenopausal women. The metabolic bone turnover is regulated with many systemic hormones and local growth factors. Additional influences include nutrition (especially calcium) and exercises.

Osteoporosis is an asimptomatic disease in a long term. Patients usually have pain in thoracal area as a first symptom of osteoporosis, exhibited in a later process of bone resorption. Hyperkyphosis in thoracic area will be exhibited 10 to 15 years after menopausal period. Impairment of chest wall function will be reduced vital capacity. Type I osteoporosis has been applied to vertebral crush fracture and fracture of distal radius, where as type II osteoprosis has been used for hip fracture.

Radiological examinations are the most available diagnostic method in diagnosis of osteoporosis and its complications, although the first changes become visible after 30% loss of bone structure.Quantitative computerised tomography (QCT) can be used to assess true bone density and has also been used to measure trabecular bone density. Ultrasonography as a method measured bone mineral density accounting ultrasound attenuation or measured speed of sound. DEXA is a gold standard for measure bone mineral density. Clinical approach has been done on lumbal vertebra or on hip.

Thyroid hormones can stimulate bone resorption directly trough thyroid receptors on bone cells.It is now belived that TSH has direct influence on bone mineral remodeling. Even a 50% reduction in TSHR expression produces significant bone loss. The replacement and mildly suppresive doses of L-thyroxine are not associated with significant bone mineral loss. Patients receiving L-thyroxin replacement therapy with low serum levels of TSH have, despite lower bone mineral densities than patients with normal TSH, no evidence of an increased fracture rate. Patients, during the first year L-thyroxin replacement therapy of overt hypothyreosis have a significant cortical bone loss.

We must insist on optimal supstitution. In a high-risk group, who need thyroid hormone replacement therapy, the risk of fracture may be prevented with estrogen replacement therapy, adequate calcium supplement and exercise and eventual medical prevention (bisphosphonates antiresorptive therapy)of osteoporosis.

Key words: osteoporosis, hypothyreosis, L-thyroxine, TSH.