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Osteoporosis in Chronic Obstructive Pulmonary Disease

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Editorial

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Abbreviations

PTH: Parathyroid Hormone; COPD: Chronic Obstructive Pulmonary Disease; RANKL: Receptor Activator of Nuclear Factor-κB Ligand; OPG: Osteoprotegerin.

Editorial

The common risk factors for Chronic Obstructive Pulmonary Disease (COPD) and Osteoporosis are age, smoking and inactivity. Destruction of bone in osteoporosis is aggravated by usage of systematic corticosteroids, Vitamin D deficiency, COPD related systematic inflammation. Morbidity and mortality is increased by impaired mobility due to fragility fractures caused by Osteoporosis. Enhanced exacerbations or reduced pulmonary function is caused by vertebral compression fractures and rib carriage fractures in patients with COPD. Bone mineral density, history of fragility fractures, population- specific clinical factors are considered in fracture risk assessment tools such as FRAX should be used for early prevention and treatment of osteoporosis in COPD. The guidelines of treatment of osteoporosis should be followed vigorously as per the intervention studies focusing on the bone in COPD.

Bone is generally classified into Cortical and Trabecular bone. Dense and strong bone found in the shaft of long bones is called cortical bone. Bone found in the interior of vertebrae and flat bones, typically occurs at the ends of long bones which is more porous and weak is called Trabecular bone. Approximately 3% cortical bone and 25% trabecular bone is replaced every year as part of continuous bone tissue renewal throughout the life. Mean annual bone loss of 0.5% to 1% results at the age of 25 to 30 years after reaching peak bone mass due to remodeling associated with an imbalance between formation and resorption, which differs by site, age, skeletal and sex. Parathyroid hormone (PTH), sex hormones, Vitamin D are the key determinants of the rate of bone remodeling and bone loss [1,2].

Osteocytes, osteoclasts and osteoblasts are complex interplay which work together at the cellular level for bone remodeling. Osteoblast forms osteoid protein matrix that subsequently mineralizes to replace bone whereas osteoclasts reabsorb bone. For mechanical stimuli osteocytes and their canicular network serve as sensors to adjust bone response. Receptor activator of nuclear factor-κB ligand (RANKL) constitutively expressed on the surface of osteoblasts. When binding to its receptor (receptor activator of nuclear factor-κB [RANK]) on the surface of preosteoclast cells, the latter differentiate into mature and activated osteoclasts. Physiologic regulator of bone turnover osteoprotegerin (OPG) a soluble decoy receptor secreted by stromal cells additionally to osteoblasts which blocks the RANK/RANKL interaction [3]. The major cause of osteoporosis is excessive activity of osteoclasts due to imbalance between RANKL and OPG [4]. The Wnt/β-catenin signaling cascade downstream of a number of osteoblast-activating proteins and receptors. Wnt signaling activates osteoblasts and bone formation, whereas reduced Wnt signaling may lead to osteoporosis [5]. In COPD patients (vitamin D deficiency, use of corticosteroids and systemic inflammation) several factors that have often been described clearly interact with these pathways.

The treatment includes the optimization of vitamin D status, the limitation of corticosteroid use, and the substitution of sex steroids.

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