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Perspective

Effects of Abaloparatide Drug on Osteoporosis

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DESCRIPTION

Osteoporosis is a prevalent, debilitating condition characterized by weakened bone structure and mass, which causes bone fragility. More than 10.2 million Americans were assumed to have osteoporosis, and 43.4 million more are expected to have osteopenia. 30% of women and 16% of men over 50 have osteoporosis, respectively. Over 2 million fractures associated with osteoporosis occur each year, with the hip, wrist, and thoracolumbar spine being the most frequently fractured bones in persons with the condition. In addition, 20% of osteoporotic patients will suffer from a spinal compression fracture. Osteoporosis and osteoporotic fragility fractures are linked to serious side effects, such as a decline in life expectancy, a loss of independence, a higher risk of future fractures, and a lower quality of life.

The standard first-line treatments for osteoporosis are antiresorptive drugs such as bisphosphonates (and less frequently denosumab). To a greater extent than conventional antiresorptive therapy, novel anabolic drugs have been demonstrated to increase bone mass and morphology as well as lower the risk of fracture. The most popular anabolic osteoporosis drug, teriparatide (human recombinant Parathyroid Hormone (PTH) 1–34), was the first in its class to receive approval in the United States and has demonstrated a considerable advantage over conventional antiresorptive therapy. The second medication in this class, abaloparatide (synthetic Parathyroid-related Peptide (PTHrP), Tymlos), has just lately become accessible for use.

A synthetic 34-amino acid peptide counterpart of PTHrP is called abaloparatide. Almost all human tissues express PTHrP, a protein with comparable properties to PTH that serves a variety of regulatory functions. PTHrP is related with humoral hypercalcemia in malignancy, which makes it detectable in malignancy even if it is not typically detectable under physiologic settings (apart from during pregnancy and breastfeeding).

PTH and PTHrP influence bone turnover *via* the Receptor Activator of Nuclear factor Kappa-Ligand (RANKL) pathway in osteoblasts. This pathway involves the stimulation of PTH and PTHrP receptors in osteoblasts, which results in the activation of cAMP, osteoblastic bone production, and later the activation of osteoclasts. PTHrP and PTH, however, are activated in different ways. The RANKL pathway is activated by at least two PTH receptor conformations, R0 and RG. Teriparatide's main target is the G-protein-independent receptor R0 (Forteo). The R0 receptor has a strong affinity for PTH and causes sustained binding and activation, which results in an initial burst of bone growth followed by bone resorption due to osteoclast activation downstream. RG, a G-protein-dependent receptor that abaloparatide targets, on the other hand, binds to PTHrP reversibly and strongly, causing a brief activation that promotes early bone formation while restricting late bone resorption and osteoclast differentiation.

The FDA has currently approved the use of abaloparatide to treat osteoporotic postmenopausal women who are at high risk of fracture. Patients who meet the "Risk" criteria for abaloparatide use may have a history of osteoporotic fracture, have several risk factors for fracture, or have tried and failed to respond to conventional osteoporosis treatments. A subcutaneous injection of 80 micrograms each day for up to 18 months is the normal dose schedule. It is advised to replenish calcium and vitamin D before starting the therapy. Abaloparatide is metabolized in the kidneys, so patients with renal impairment may require a different dose.

A second-generation anabolic drug called abaloparatide is used to treat osteoporosis. It differs from teriparatide, a first-generation anabolic therapy, in that it binds to the RG configuration of the PTH receptor with high affinity and is reversible, causing transient osteoblast activation that maximizes initial bone formation while restricting late bone resorption and osteoclast differentiation. Studies on both animals and humans have demonstrated the positive effects on BMD, bone morphology, and fracture protection. The relatively low side effect profiles of aloparatide therapy, as well as its low cost, are additional benefits.

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