使用生物製劑治療骨質疏鬆

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Osteoporosis is a skeletal disease associated with an imbalance between formation and resorption of bone which leads to loss of bone mass, fragile bone structure and finally bone fracture. Although it was considered to be a disease of old female, but evidence showed that many drugs and genetic traits predisposed patients to osteoporosis.

Recently ,in addition to those approved for the treatment of osteoporosis such as bisphosphonate, endocrine replacement , the discovery of the wingless-type (Wnt) signaling pathway, sclerostin produced by osteocytes and the Dickkopf (DKK1) protein secreted in bone paved the way to the development of novel therapy of osteoporosis. Parathyroid hormone PTH 1–34 (teriparatide) stimulates bone formation through inhibition of sclerostin, DKK1, and frizzled protein; increases BMD; improves microarchitecture; and decreases fractures and is approved for osteoporosis. Denosumab, a human monoclonal antibody that specifically binds RANKL, markedly reducing bone resorption, increases bone density, and reduces fractures and is approved for osteoporosis. The anti-sclerostin antibodies (romosozumab, blosozumab) increase bone mass by neutralizing the negative effects of sclerostin on the Wnt signaling pathway are now under evaluation in clinical trials. In a phase 2 study, in postmenopausal women with low bone mass, romosozumab was associated with increased bone mineral density and bone formation and with decreased bone resorption. In another phase 2 study, Blosozumab treatment resulted in statistically significant dose-related increases in spine, femoral neck, and total hip BMD as compared with placebo. In the highest dose group, BMD increases from baseline reached 17.7% at the spine, and 6.2% at the total hip. In the near future, more and more biologic agents will be evaluated in the clinical trials base on the novel molecules found in the regulation of bone homeostasis.