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Commentary

Basic Treatment of Musculoskeletal Disorders

Ronald George*

Department of Nursing, Benha University, Benha, Egypt

DESCRIPTION

The most promising new therapeutic target for the treatment of osteoporosis and osteoporotic fractures is a bone anabolic therapy. Parathyroid hormone 1-84 (PTH) and its biologically active 34-residue amino-terminal fragment, known as teriparatide, are the only substances currently used in osteoanabolic therapy (PTH). These PTH molecules are osteoanabolic when given intermittently (once per day). PTH has certain drawbacks, too, like the demand for daily self-injections, high cost, need for refrigeration, 2-year usage limit, and boxed warning enforced by the US FDA about osteosarcoma in rats in preclinical toxicity tests.

Additionally, the rise in bone resorption that commonly follows the increase in bone production seen with PTH treatment causes a 'unwanted' increase in bone remodelling. Which, in contrast to PTH, are linked to a decrease in bone resorption, making them much preferred. Blosozumab, a humanised monoclonal antibody targeted against sclerostin, was studied in postmenopausal women with low Bone Mineral Density (BMD) in a randomized, double-blind, placebo-controlled multicenter clinical trial. Blosozumab injections for a year had significant anabolic effects on the skeleton and were well tolerated.

There is currently no cure for osteoporosis that has already developed, and the duration of all antiresorptive treatments is constrained. For the majority of people, early intervention can stop osteoporosis. Medical treatment can prevent the progression of osteoporosis in those who already have it. If secondary osteoporosis is evident, the underlying condition should be treated. The risks and advantages of treatment should be discussed between the clinician and patient, and therapy should be tailored to each patient's unique clinical situation. The American College of Physicians' clinical practice recommendation states that therapy is focused on fracture prevention because osteoporotic fractures are linked with severe disability, morbidity, death, and financial costs.

Additionally, treatment guidelines for osteoporosis are available from the American Association of Clinical Endocrinologists and a recent collaboration between the European Society for Clinical and Economic Aspects of Osteoporosis and Osteoarthritis and the International Osteoporosis Foundation. Preventive measures include changing general lifestyle factors, like increasing weight-bearing and muscle-strengthening exercise, which have been linked to fractures in epidemiologic studies, as well as making sure you get the recommended amounts of calcium and vitamin D as a support for active ant fracture therapy. Adequate calcium, vitamin D, and ant osteoporotic medications such bisphosphonates, the RANKL inhibitor denosumab, parathyroid hormone, raloxifene, strontium ranelate, and, until recently, oestrogen are all part of medical therapy.

The mainstay of osteoporosis treatment is bisphosphonates, which have been shown to be effective in lowering the risk of fracture over three to five years of treatment by a large number of placebo-controlled trials. Despite the fact that bisphosphonates are often safe and well tolerated, concerns have been raised about potential side effects from prolonged use. Typical Femoral Fractures (TFF), osteonecrosis of the jaw, and oesophageal cancer are specifically related with continuing use of bisphosphonates after 5 years. The prevalence of ONJ is highest in oncology patients (1–15%), when high doses of these drugs are frequently administered. On the other hand, the incidence of ONJ in the community of people with osteoporosis is estimated to be between 0.001% and 0.01%, which is somewhat higher than the incidence in the general population (0.001%).

Recently, ONJ was discovered in patients receiving Dmab who had never taken bisphosphonates, necessitating the inclusion of Dmab in the criteria. A causal link between the use of bisphosphonates or Dmab treatment and ONJ has not been demonstrated, despite the likelihood of such a connection. Another issue is that, despite variations in the strength of linkages and magnitudes of effects, studies with radiographic evaluation consistently find strong correlations between the use of bisphosphonates and AFFs. Patients on bisphosphonates have a modest absolute risk of AFFs, with cases per 100,000 person-years ranging from 3.2 to 50. Long-term use, however, might be linked to a larger risk.

Correspondence to: Ronald George, Department of Nursing, Benha University, Benha, Egypt, E-mail: Ronaldg@gmail.com

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Bisphosphonates seem to concentrate in stress fracture-prone regions. The methods through which stress fractures typically heal could be hampered if targeted intracortical remodelling at the site of an AFF were suppressed. This theory's evidence suggests that the chance of an AFF may decrease when bisphosphonates are stopped.

The femoral neck and lumbar spine's bone mineral density were maintained, but there was no consistently lower fracture rate, according to an analysis of trials where bisphosphonates had been used for at least three years and for which fracture data had been obtained. In light of all of these discoveries, the FDA updated its advice for using these medications three to five years later.

It is appropriate to think about taking a "drug holiday" because bisphosphonates accumulate in bone with some enduring antifracture efficacy when medication is ceased. There is much debate on the ideal length of therapy and vacation, both of which should be determined by each person's judgement of risk and reward. For long-term management of osteoporosis patients, replacing potent suppressors of bone remodelling, such as bisphosphonates or the RANKL inhibitor Dmab, with less potently active medications may become an appealing alternative to simply stopping treatment and subjecting patients to an increased fracture risk.