

Loss of Bone Mass or a Case of Misdiagnosing? - Case Report

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Abstract

DXA is a convenient, safe, accessible, affordable method, and it is the golden-standard tool to access bone mass. Teriparatide is an anabolic agent that activates bone remodeling and improves bone microarchitecture, but its action is complex and varies among skeletal sites. This report is a case presentation that illustrates a considerable reduction in BMD at non-dominant forearm with a BMD improvement at lumbar spine and hips after two years of teriparatide once daily. Bone metabolism knowledge explains this intriguing result.

Keywords: Scan Interpretation; DXA Analysis; Osteoporosis; Forearm; Teriparatide; Cortical Bone; Cancellous Bone

Abbreviations

BMD: Bone Mineral Density; DXA: Central Dual-Energy X-Ray Absorptiometry; BMI: Body Mass Index; TPT: Teriparatide; PTH: Parathyroid Hormone; FDA: Food and Drug Administration

Introduction

The understanding that low bone mineral density (BMD) increases risk of fractures explains why osteoporosis became a significant public health issue, considering the disease a major risk factor for lumbar spine, forearm, and hip fracture [1]. Central dual-energy x-ray absorptiometry (DXA) is the golden-standard tool to measure and monitor BMD, and to diagnose osteoporosis as well [2].

DXA is a convenient method to the patient since it is safe (not invasive and requires a low dose of ionizing radiation), accessible and it is an affordable test. This technology is advantageous to the physicians' evaluation considering the well-standardized references. However, those DXA features do not overcome the bone metabolism and physiology knowledge required by the specialist during analyses.

The case presented here pictures a decrease in BMD at the non-dominant forearm, and an increase in lumbar spine and hip BMD in a patient with good treatment compliance.

Case Report

A 78-year-old woman presented at our DXA service for osteoporosis follow up. She is 159 cm (62,59 inches) tall and weighs 59 kg (130 pounds), with BMI = 23,33 kg/m2. Her baseline DXA showed T-scores of -2,1 at the lumbar spine, -2,6 at the total femur and -3,2 at radius 33%. After two years, followed up DXA demonstrated a 23% increase in the lumbar spine bone mineral density (BMD), 4% increase at the total hip, but a loss of 15% of the non-dominant forearm (Figure 1).

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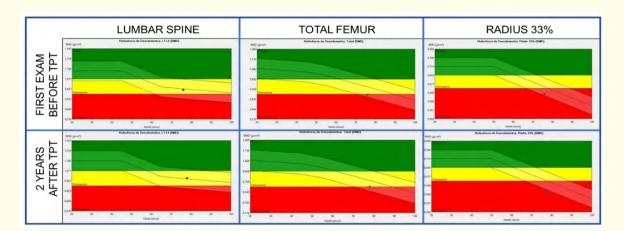


Figure 1: Comparative site and BMD analysis between first DXA, before teriparatide treatment, and DXA 2 years after teriparatide treatment. TPT: Tteriparatide.

The DXA results raised the question what could explain the bone gain at the hip and spine, but the bone loss at the forearm. The answer is in her past medical history with teriparatide use for the past two years.

Discussion

Therapy with teriparatide (TPT) is indicated as a second-line osteoporosis treatment, when bisphosphonates where ineffective, and as a primary choice if a very high risk of fracture or previous fracture is identified [2]. The drug is the amino-terminal fragment of human parathyroid hormone (PTH 1-34), an anabolic osteoporosis therapeutic agent. TPT with intermittent administration delays bone reabsorption markers expression and simultaneously stimulates markers of bone formation, activating bone remodeling and so improving bone microarchitecture [3,4].

At both cancellous and cortical bones, the response to TPT is an increase in bone formation associated with a gap in bone reabsorption. There so, anabolism exceeds bone reabsorption [5]. However, TPT anabolic action varies among skeletal sites: at cancellous sites, such as the lumbar spine, bone reabsorption is decreased, leading to an increase in trabecular volume; and, at cortical sites, such as distal 1/3 of the radius, bone reabsorption gap is shorter [4-6]. The presented case illustrates a significant reduction in BMD at non-dominant forearm with a BMD improvement at lumbar spine and hips after two years of teriparatide once daily. These intriguing results are supported by the fact that, at predominantly cortical sites, TPT promotes neocortical bone reabsorption, increasing bone porosity, as well as augmenting periosteal bone sedimentation leading to a gain in cortical thickness and bone diameter (Figure 2) [4-6]. As a result, cortical bone gets an inner endocortical porosity and a thicker cortical surface, therefore a bone with less BMD.

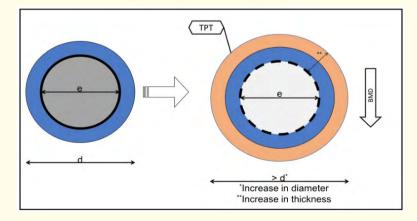


Figure 2: Graphic representation of cortical bone changes after TPT treatment: 'e' represents endocortical diameter, that has no change in size, but increased in bone porosity; 'd' represents total bone diameter, which increases due to periosteal bone sedimentation – increase in thickness. The final result is a reduced BMD, but with a thicker cortical surface.

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It is important to highlight, however, that besides the increased in endocortical porosity there is no increase in fragility. Burr and Cols [7] evaluated the influence of porosity induced by TPT on biomechanical fragility. Ovariectomized monkeys were treated with TPT for 18 months and had a dose-dependent increase in intracortical porosity, most concentrated near the endocortical surface, with no significant bone strength effect [6]. The explanation is that the endocortical surface, where the porosity was most concentrate, has a small mechanical effect. This mechanical effect, bone bending rigidity, is proportional to the bone moment of inertia divided by bone radius. Moment of inertia is a geometrical property that reflects how mass is distributed. Because bones are not symmetric and isotropic, the moment of inertia is not always at the geometric center (Figure 3). As endocortical porosity increases, the moment of inertia also increases, compensating decreased bone mass.

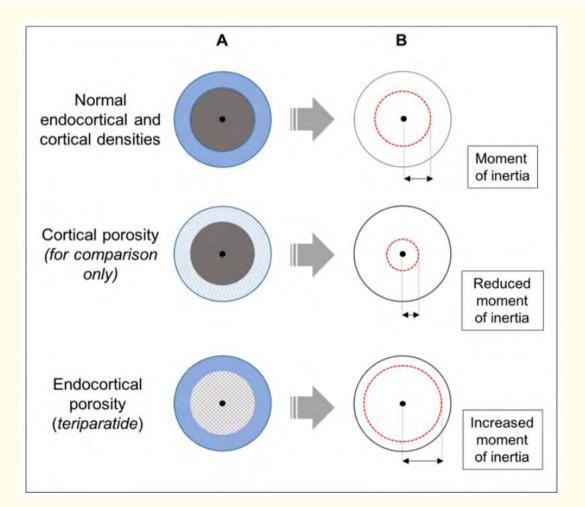


Figure 2: At column A, there are three different bone densities: normal bone, bone with cortical porosity and bone with endocortical porosity; column B, schematic illustrates the bone moment of inertia (dotted line) according to bone mass distribution. Within endocortical porosity, bone density concentrates at cortical surface and the moment of inertia increases.

Conclusion

TPT has a complex effect on the skeleton, and it still is the only anabolic bone agent approved by the Food and Drug Administration (FDA) and other. Even though DXA is the golden-standard tool for osteoporosis diagnosis and follow-up, some bone strength features,

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such as bone geometry, are not reflected. Those limitations, however, can be overcome with the physician expertise and experience by understanding bone physiology and its clinical implications. Referred physicians must be aware of DXA limitations and monitor osteoporosis improvement with a complete analysis of the exam and clinical features, and along with it approach all present risk of fractures elements.

Conflict of Interest

We have no conflict of interest to declare.

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