

Osteoporosis – time to downgrade?

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The history of osteoporosis started in the early 1990s. In Poland, it was a time of “Sturm und Drang”; numerous osteoporosis centers emerged, equipped with dual-energy X-ray absorptiometry (DEXA, currently DXA) or ultrasound machines and produced results that were difficult to compare. Introduction of bisphosphonates, educational activities for physicians and massive PR campaigns sponsored by the pharmaceutical industry made osteoporosis a major focus of public health. Headlines such as “osteoporosis – the silent bone killer” and DXA results presenting bone loss as a percentage of normal bone mineral density (BMD) created a kind of “osteoporotic hysteria”.

Clearly, the 1994 WHO diagnostic criteria gave boost to the fulminant development of the osteoporosis concept [1]. According to WHO experts, osteoporosis has operationally been defined on the basis of DXA; osteoporosis was diagnosed if BMD lay 2.5 standard deviations (SD) or more below the average value for young healthy individuals (T -score of < -2.5 SD). Subjects with DXA results not lower than -1 were regarded as normal, those with the results in between as having osteopenia [2]. The criteria justified use of DXA machines for making the diagnosis. Moreover, clinical trials in osteoporosis usually required low BMD as an inclusion and drugs were licensed for use in patients below a given BMD threshold. Clearly, the WHO definition perfectly suited the needs of industry. But did it correctly describe the clinical problem?

The T -score cut-off of -2.5 separates subjects suffering from osteoporosis (presumably a “real illness”) from those with osteopenia (a “pre-disease” state). For most chronic diseases, there is a population of subjects at risk; some of them develop symptomatic disease (such as ischemic heart disease), and some of those who have developed a disease suffer complications (such as chronic cardiac failure). For osteoporosis, there is a population at risk (subjects with osteopenia, no clinical symptoms), some patients develop “disease” (osteoporosis,

no clinical symptoms), and some develop complications (bone fractures, seen both in osteopenia and osteoporosis). In fact, there is no real qualitative difference in the osteopenia and osteoporosis group as defined by WHO; both represent the same clinical situation – a patient being at increased risk of bone fractures. The difference is quantitative; most (not all) patients with a “disease” osteoporosis represent higher risk compared with the osteopenic ones. So, what is the reason to label the first “a disease”? There is an explanation; the risk situation requires at most some preventive measures, risk can rationally be assessed even by the patients themselves. The disease requires treatment – most patients “suffering” from osteoporosis want to be treated before the disease “kills their bones”.

The current concept of osteoporosis often leads to paradoxical situations. Patients with T -score results near the -2.5 cut-off sometimes repeat DXA measurements on different occasions and using different DXA machines. Some results are above and some below the -2.5 T -score threshold. This means that at the same time the patient has and does not have osteoporosis. One cannot verify the diagnosis as there are no clinical symptoms and no additional confirmatory tests available. On the other hand, patients with advanced osteoporosis and concomitant osteoarthritis will often have normal or even high T -score results. The current concept of osteoporosis is an artificial construct purely based on statistical considerations. In the statistician’s mind, a value that lies on the Gaussian curve at least ± 2.5 standard deviations from the average is so uncommon that it surely represents an abnormality, a “disease”. Following this idea, artificial “disease constructs” can be created for various medical variables, even if there is no rationale substantiating a new entity. This is absurd and, in my opinion, this applies to the current concept of osteoporosis; part of the entire fracture risk spectrum is taken out in an arbitrary manner and called a “disease”, although there are neither clinical symp-

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toms nor additional laboratory abnormalities justifying a clinical entity.

The fracture risk is the real problem and should be focused on. Two years ago I was asked by Prof. Piotr Głuszko to moderate the Workshop on New 2013 Guidelines in Osteoporosis held on May 25th, 2013 in Poznań. I listed all my doubts about the disease and took a look at FRAX, the well-known web-based fracture calculator [3]. Its Polish version started on June 1st, 2011. The FRAX algorithms calculate the 10-year probability of hip fracture/major osteoporotic fracture. A counter on the website shows the actual number of assessments made. I looked at the counter on May 22nd, 2013 and it was 50767, two days later it was 51026, and the average daily number of assessments since the start of the website was 70.5. Assuming the estimated number of patients with confirmed osteoporosis in Poland (about 2.8 million), it gave 0.018 assessments per patient (at most, the risk was assessed in 18 out of 1000 patients). I was writing this paper on June, 7th, 2015 and the counter showed 2 927 523 assessments since June 1st, 2011 (1995 assessments per day, on average), which is great progress. It seems that more and more physicians perceive the problem from the perspective of fracture risk, not as a certain BMD threshold. Of course, FRAX is not the ideal tool: the risk cannot be calculated for patients aged less than 40 years; it does not contain all risk factors (such as falls); and it may overestimate fracture probability in patients with the *T*-score for the lumbar spine greatly exceeding that for the femoral neck.

In their book, Dr G. Welch and his colleagues critically deal with osteoporosis as a medical concept [4]. The authors analyzed data available on treatment success for decreased BMD; among the patients there were winners (treatment saved them from a fracture, about 5%), those treated “for naught” (patients who developed fracture despite treatment, 44%) and losers (treated but never would have had a fracture without treatment, 51%). These data show that the value of drug treatment for decreased BMD is limited. More attention should be paid to other measures such as regular exercise, prevention of falls, changes in diet/lifestyle, adjustment of home environment, and regular examination of sight/hearing, with the correction of underlying problems. The patients may profit more from these simple measures than from medicines. Also, in spite of high frequency of vitamin D deficiency in Europe, measurements of serum 25-OH-D should become routine [5].

I believe the time has come to downgrade osteoporosis from its status as a “disease” and start to talk about a high fracture risk population. We should stop stigmatizing our patients with the label of a potentially

lethal “disease”. Instead, we should look at the risk and discuss with the patients how to eliminate risk factors, patients’ expectations, and acceptable treatment options. It is time for a “treat-to-target” approach, whereas the goal is a reduction of fracture risk to a level that is satisfactory for both the physician and the patient, based on their shared decision [6]. Should we be happy every time we manage to get the *T*-score above the -2.5 threshold? Or should we aim at significant reduction of fracture risk instead of “curing” BMD?

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