Osteoporosis and Reduction of Bone Mass by Age and Sex

Healthcare

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Dorina Ruçi | **Mother Teresa University Hospital, Tirana, Albania.**

Abstract

The bone is a living tissue constantly changing. From the moment of birth until the age of adulthood bone tissue grows, develops and strengthens. This process reached its maximum in the third decade of life known as peak bone mass. Over the years bone mass falters through resorption while new bone tissue replaces the previous one. This process is known as remodeling. In osteoporosis the process of resorption exceeds that of replacment with the end of mass reduction and weakening of bone quality with increased propensity for fractures. Types of bone loss by age and sex were studied in 287 normal subjects (205 women and 82 men) from age 20 to 78 years old and 176 women and 39 men with Colles fractures and colli humeri with osteoporotic nature. Bone mineral density was measured by DEXA Lunar GE. In normal women the reduction of bone mass starts at a young age and is linear. Loss of bone mass is noted at age 50 and it is noted up to 65, falling after age 65. Total loss of bone mass resulting 47%. In normal men reduction in bone mass is smaller. Bone mineral density in patients with osteoporosis was significantly lower than that in normal subjects in relation to age and sex.

Introduction

Osteoporosis is a metabolic bone disease characterized by a decrease in bone mass and bone damage microarchitecture leading to decrease of mechanical strength of bone tissue with increased risk for fractures.

A distinctive feature of osteoporosis is a report stored between mineral and matrix content of collagen that distinguishes from osteomalacia and rickets in which the decrease of mineral mass is seen having the quantity of collagen unchanged.

Osteoporosis is the most common metabolic disease of bone in developed countries. In fact, osteoporosis is a disease that starts with a quantitative change (loss of bone mass) and the deepening of disease changes take a qualitative shape (substantial changes to bone microarchitecture).

The inevitable result of this combination is to reduce the mechanical resistance and increase the possibilities for bone fractures under the action of a no greater traumatic force. Part of clinical researchers include in the clinic syndrome of osteoporosis fractures, and osteopenine as asymptomatic stage of the disease during which no fractures occur.

Similarly, can be taken as an example the arterial hypertension as a preceding stage of the cerebral vascular accident. Osteoporosis is fundamentally a reduction of osseous tissue mass while its qualitative reports do not change.

However, this definition today has quite unclear sides which could not explain the different clinical manifestations of osteoporotic fractures. It is often noticed that the reduction of bone mass does not have a proportional increase in vertebral fractures, or on the other side are observed osteoporotic fractures even when the bone measure is not necessarily reduced. For the same amount of bone mass seems to play a role also the internal mechanics of the bone itself.

It is seen that the mechanical resistance of bone depends not only on the density of bone tissue but also by other parameters studied as crystals hydroxyapatite (HA) size, placement and management of their crystals, the ratio between formed crystals and uncrystalized yet amorphous mass, size and orientation of fibers of Collagen.

Other authors have noticed that the skeleton size has an independent role of bone density. People with large skeleton suffer less osteoporotic fractures. In conclusion, we can say that based on today's data osteoporosis is not only a quantitative disease but but it is a qualitative disorder of bone tissue that has connections with mechanoceptors and the internal mechanics of bone. But until today in the clinical practice can be tested only bone density with different handsets and non-invasive techniques.

Heterogeneity

Due to multifunctional etiology it is not surprising that osteoporosis is a heterogenious disorder. Relative contribution of age estrogenic deficit is mentioned quite a lot in the past, but it is difficult to differentiate these two factors in most of the patients. It is even more apparent that there are other etiological factors including those that are predominantly unknown. Predominant etiology may differ from one patient to another. The main contribution of the peak bone mass to the risk of future fractures shows that the risk factors that appear over the years, childhood and adolescence may contribute as much as aging and menopause on risk fractures. Etiological factors that could be treated and prevented have a great importance for the clinic as well as for the public's health, especially if they are common, as it is with low bone density.

How should we understand osteoporotic fracture?

Besides clinical and socio-economic consequences that fractures bring, it has another crucial implication of this type of fractures in each special patient. The presence of a fracture itself is a risk factor for other potential fractures in the future, regardless of bone density. The explanation for it is unclear to the vertebrae, it can be explained partly by changing the distribution of cargo in neighboring vrtebrae troops. This is why vertebral fractures tend to occur in the area of high thoractic and lumbar border.

Mechanisms: bone loss in postmenopausal women occurs as a result of the increased bone remodeling and imbalance between activity of osteoclasts and osteoblasts. Bone remodeling occurs at certain points within the skeleton and progresses in an orderly way with bone resorption which is always followed by bone formation, a phenomenon known as coupling. In cortical and spongioze bones the sequence is similar. Bone size which is fallen silent promotes activity (beginning) and osteoclasts begin to resorb the bone (progression) by forming a cone cut (cortical bone) or a tranches (spongioze bones). Osteoblasts form bone matrix which continuously mineralizes. This process requires about 8 months (Eriksenet al 1984). If resorption and boneformation processes are not balanced, then the remodeling imbalance occurs. In postmenopause it is noted this imbalance by increasing the frequency scale of remodeling cycles (activation frequency).

The remodeling imbalance leads to irreversible bone loss. There are two other reasons for irreversible bone loss which were recognized as modeling errors. The first is the erosion of extremely large spaces in cortical bone haversian system. Refilling resort regulated by signals coming from more peripheric osteocites is greater than 90μm. Increased diameters of cavities cause haversiane channels to enlarge which being added through age lead to increase of porosity of cortical bone. Similarly osteoclastc penetrate in trabecular plakes and remove the matrix tablets that need osteoblastic replacement of the resorbed bone. In both ways, errors of modeling affect the reduction of density as well as the corticales spongiozes damaging structural integrity.

Causes

1. Estrogenic deficit: Menopause occurs around the age of 50 in a normal woman. Reduction of ovarious hormone production is more gradual and begins several years ahead from the last period. The first changes of bone mass and calcium metabolism is observed during the perimenopausal transition. Ovarian estrogen is the hormone that plays a pivotal role in mineral metabolism, without forgetting the influence of progesterone and ovarian androgenes. In pre-menopausal stage there is no apparent loss of bone mass and fractures may occur in any part of the skeleton. The most obvious effect of menopausal skeleton is the increase in the incidence of fractures, that for forearm and vertebrae is quite evident in the first decade after menopause. This effect is attributed to rapid reduction of bone mass, especially of that spongioze.

2. Effects on calcium metabolism: bone loss after menopause is associated with negative changes in calcium balance, which is partly due to reduction of intestinal absorption and increased urinary loss. Reduction of intestinal absorption is accompanied by reduction of active vitamin D3, suggesting that estrogen affects the synthesis of protein that transports vitamin D3. In kidneys tubular reabsorbtion of calcium increases in the presence of estrogen, stimulating parathyroid hormone. Finally the effects of menopause on the skeleton are such that promote recycling but rather bone reabsorbtion. This bone loss continues for the whole postmenopause, leading to possible fractures increase. Bone loss in post menopause occurs in two stages: In the first stage there is an accelerated bone loss that continues for 5 years. Following, the bone loss is gradual and waning. This second men's stage is slow and starts at the age of 55. Circulating estradiol levels are reduced by 90% during menopause. This bone loss can be prevented by giving estrogen and progesterone. It is estimated that this phase of rapid loss contributes to 50% of total bone loss in the vertebrae of women.

3. Aging: slow phase of bone loss is attributed to age-related factors such as the growth of the level of parathormone and aging of osteoblasts. An increase in the level of parathormone is seen in both men and women over age. Parathormone levels correlate with biochemical markers of bone cycle, and that both can return to the values of a new adult after intravenous calcium perfusions. Increase of parathormone is due to the decrease of renal and intensinal calcium absorption. The latter may come from the deficit of vitamin D. Regardless of the cause a calcium-rich diet restores parathormone values near normal values.

Patients and Methods

Normal subjects and patients

287 normal patients (205 women and 82 men) were weighed for bone density. Age varies from 20 to 78 years. All patients presented no pathology, ambulatory healthy and drug-free therapy history. None of them presented lumbago or history of fractures. All patients in the column x-rays show no fracture data for earlier or new or more advanced osteoarthritis.

215 patients (176 women and 39 men) with osteoporosis confirmed by bone fractures fragility (minimal trauma) in the wrist and neck of humerus were taken in the study. The average age was 57.3 years (50-75 years). All the patients had x-rays and a column with one or more mainly spontaneous comprehensible vertebral fractures. All the patients were patients without serious medical problems, no history of drug for other problems excluding osteoporosis.

Bone Densitometria

Bone mineral density was determined by DEXA technique with the same GE Lunar camera, and by the same technical examiner.

Statistical Methods

Regression of bone density measurement was conducted in two ways. First, linear regression was performed separately for ages 20-50 years, and 51-65 years, ≥ 51 years and ≥ 66 years respectively. Linear regression results in different age groups were compared for evaluation in connection with several variables such as age, length, weight.

Results

Variable with the greatest effect was in aged women as well as in men.Variable of age and weight taken together affect 71% of women and 65% of men. Because they are two variables that define normal lomits in men, it is unclear whether correlation should be done according to age. Adding length as a variable had no effect in males, while the difference was small, while the difference was small but statistically sensitive in women. Body weight resulted in a small but receptive change, in the reduction of the residual variance in both sexes.

Normal Subjects

Loss of bone mass occurs to all women. CPD age regression in femoral bone was linear and with an intensity 0.0092 g / cm ² per year. The total estimated loss to age 90 was 47% less than the predicted loss for the age 20. Regressive analysis shows a simple linear function for all ages. It wasn't noticed any accelerating decrease (curvilinear) of density in postmenopausal period between 51-65 years. The potential effect of menopause on bone loss accelerates further studies by changing the correct age when menopause began, in these subjects. For this analysis subjects in pre-menopause were studied for their age, while in postmenopause subjects were studied by calculating the age of 50 plus the number of years after menopause. No major difference was observed (accelerated growth of bone loss) in postmenopausal (Table 1). From age 51 to 65 years, DMK is dropping at a rate 0.0118 g / cm ² / year; after age 65 the reduction of bone mass is lower (Table 3).

Table 1. *DEXA koksofemorae regarding menopause in normal women (According to linear regression analysis)*

Menopause	A	B	S y-x
Total	1.5364	-0.0080 *	0.1640
Age 20-50 (premenopause)	1.5596	-0.0077 §	0.1495
51+ (postmenopause)	1.2705	-0.0043 §	1.1645

*P < 0.001 §, P < 0.05

Table 2. *Parameters of linear regression of age in normal women*

g/cm²	N	A	B	Sy-x
Total	102	1.5898	-0.0092*	0.1468
Age 20-50	42	1.5706	-0.0083*	0.1549
Age 51 +	63	1.4146	-0.0067*	0.1412
Age 51-65	24	1.5958	-0.0099	0.1523
Age 66 +	39	1.4339	-0.0069	0.1375

y= A+ B. Age (years) * P<0.001 Tab 4

Table 3. *Parameters of linear regression of age in normal men.*

g/cm²	N	A	B	Sx-y
Total	82	1.3299	-0.0021*	0.1595
Age 20-50	39	1.4076	-0.0044	0.1761
Age 51 +	43	1.2531	-0.0010	0.1456
Age 51-65 vjeç	17	1.9535	-0.0133	0.1543
Age 66 +	26	1.0450	0.0018	0.1382

y= A+ B. Age(years) * P < 0.05

Discussions

We found significant changes in the loss of bone mass in normal patients from patients with osteoporotic fractures in the same age group. We saw that normal women have little loss of bone mass until age 50. Bone loss accelerates from 51-65 years and then reaches a plateau sloping moderately after age 65 years.

In women with osteoporotic fractures we found an increased bone loss in 50 years of age almost at the same rate as the age group of 51-65 years. In addition, these data suggest that except the menopause to the appearance of clinical osteoporosis affect and other important factors that do not depend on menopause and primarily related to modifiable or non-modifiable factors (genetics) that determine since in adulthood (25-30 years) peak of the bone mass for each individual. Among these factors we can mention getting adequate amounts of calcium, protein, vitamin C, vitamin D, micronutrients, physical activity, smoking, coffee, different medications, early appearance of menstruation, over or body underweight, etc.

We found that the cumulative reduction of CPD spongiotic bone from early adult age until deep old age for women was 47% and 14% for men.

Starting from the reports of Albright et al, added loss of bone mass is suggested as the main factor determining osteoporosis. Newton-John and Morgan proposed another hypothesis according to which bone loss due to aging is a constant phenomenon in all people, so that the main factor whether or not appears the clinical osteoporosis, it is the peak of bone mass in the adult (after skeletal maturity). These two models are not mutually exclusive and each hypothesis taken alone does not explain the patogenesis of osteoporosis.

Smith et al found that bone loss depending on age was an exponential function of bone mass by proving that subjects with greater bone mass and speed have greater reduction of bone mass.

The data obtained allow us to support the theory of Newton-John and Morgan under which a maximum of reduced bone mass at skeletal maturity is an independent risk factor for significant mass loss.

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