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Osteoporosis

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By Bhavini Patel

Dekalb, Illinois

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Student Name: Bhavini Patel

Faculty Supervisor: Dr. Nancy Nuzzo

Faculty Approval Signature:

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ABSTRACT:

This paper examines the effects, causes, symptoms, prevention methods, and treatments of osteoporosis. Various forms of literature including journal articles and books were used to research this topic. The information gathered was compacted into a paper that explained and stated the most relevant and proven facts regarding osteoporosis. The information was then compacted further when a pamphlet was made consisting of the most important information. The purpose of the paper was to provide a tool for the college age population to help prevent a common disease that could affect them later in life. The pamphlet was assembled to be a quick reference guide of the most important information and to spread the information more quickly and easily. However, because of technical dilemmas, the pamphlet was unable to be utilized in the manner that was planned but could be used in a clinical situation.

Bhavini Patel

Osteoporosis

Osteoporosis is a disease that affects over 28 million people. Half of the individuals with osteoporosis and its effects are unable to walk unassisted and 25% are confined to long-term care in a nursing home. [13] Osteoporotic bone loss is responsible for up to 1.5 million fractures a year.. Osteoporosis means "porous bone." Its effects can be potentially devastating for both the patients and their family. There is a pathological process that characterizes the disease. Many other diseases increase the occurrence of the pathological process. In fact, osteoporosis is a secondary condition to many diseases. It occurs as a result of the effects of other diseases. Lifestyle related causes also increase the occurrence. There are also idiopathic causes. Once osteoporosis is suspected, there are many diagnostic methods to confirm it. Once it has been diagnosed, there are currently many treatment options available. An overview of osteoporosis begins with its characterization and prevalence.

The disease is characterized by low bone mass and thinning of bone tissue. Bone is made up of cortical bone and trabecular bone. Cortical bone is the dominant type of bone found in the shafts of long bones. Trabecular bone on the other hand is mostly found in vertebrae, pelvic bone, ends of

bone on the other hand is mostly found in vertebrae, pelvic bone, ends of long bones, and other t1at bones. Trabecular bone is more metabolically active than cortical bone. This may be because of its greater surface area. Trabecular bone is therefore more responsive and affected by changes in minerals.

As a person develops, their bone develops to a level called the peak bone mass, it is the maximum mass of bone that the body is going to reach. After this stage is reached, the slow phase of bone loss begins. **It** begins around age 40 for cortical bone and 5-10 years earlier for trabecular bone. **In** women a "transient accelerated postmenopausal" phase of bone loss also occurs. This process occurs due to estrogen deficiency that accompanies menopause. It results in a disproportionate loss of trabecular bone as compared to cortical bone. On average, 25% is lost in both the trabecular and cortical compartment for both men and women. However, post-menopausal women loose an additional 10% from the cortical compartment and an additional 25% from the trabecular compartment around the age of 40. This additional loss increases as women get older.. Therefore, overall, women loose 35% from cortical bone and 50% from trabecular bone. Evista, Fasomax, and hormone replacement therapy are some of the recent more

common drugs that are used to decrease bone loss in women that occur as a result of estrogen deficiency. [13]

Bone remodeling is also affected by age. Bone formation and resorption occurs in bone remodeling units. In the remodeling cycle, the first thing that occurs is that the flattened lining cells are activated. They retract and expose the underlying bone and recruitment of the osteoclast precursor cells occurs. The osteoclasts (bone destroying cells) then migrate toward the expose area of the bone. During the following 1-3 weeks, the cells form a tunnel in the cortical bone or a lacuna on the surface of trabecular bone. The osteoclasts then disappear and are replaced by osteoblasts (bone forming cells). The osteoblasts fill in the "holes" during a period of 3-4 months. These changes occur all throughout life. Osteoporosis occurs as a result of an imbalance at each bone-remodeling unit. Imbalance starts to occur around the age of 40, same as when the slow phase of bone loss begins. An increase in the number of bone remodeling units leads to an increase of bone loss. Bone turnover time (the time it takes for bone to break down and rebuild itself) is slowed down.[10] In the slow phase of bone loss, the osteoblasts fail to completely fill in the "holes" formed by the osteoclasts. This leads to thinning of trabecular bone, however, their connectivity is still present. In the accelerated

postmenopausal phase of bone loss, there is trabecular perforation and loss of the connectivity. [13] Bone density tests can be done to determine how much perforation there is. They will be discussed in detail later.

If the bone density is extremely low, bone starts to resemble thin lace (the spaces within bone become enlarged), it becomes fragile and is easily fractured. This process can continue unnoticed until a fracture occurs. In fact, fractures do not occur until bone density has fallen below values found in young adults. Normal bone density is 1.0 gm per sq em for vertebrae and the proximal femur and 0.4 gm per sq em for the ultra distal radius. The lower the level of bone density, the higher the risk of fracture. [13]

There are many things that increase chances of getting osteoporosis:

-bone mass is affected by genetic and lifestyle factors

-unbalanced diets and disordered eating

-low calorie intake and excessive exercise[12]

-women who are thin tend to have lower bone density and are at more risk for fractures.[12]

-calcium and vitamin D deficiency[12]

-estrogen deficiency[12]

-women who enter menopause at an early age are more

likely to loose more bone and women who enter menopause with lower bone mass are at a higher risk for developing osteoporosis, [12]

-Steroid use [10]

-Too much or too little thyroid medication [11,12]

-women with type I diabetes are thought of to be at higher risk for osteoporosis [12]

-Smoking interferes with the body's ability to absorb calcium [12]

-Increased alcohol intake may increase the risk for osteoporosis [10]

In fact, smoking and alcohol intake doubles the risk of developing osteoporosis. [13] The risk is significantly greater among people who drink than those who do not. [15] Young people who drink alcohol have been shown to have thinner bones than other young people of the same age who do not drink alcohol. [13] The risk is increased by a factor of 1.007 for each ounce year (1 ounce per day for a year) of cumulative exposure. The risk is also significantly greater among people who smoke than those who do not. The risk is increased by a factor of 1.009 for each pack year (1 pack per day for a year) of exposure. [15]

Obesity on the other hand, is not a factor that increases the risk for osteoporosis. In fact, it helps decrease it. The risk among obese men was less than a third of that among non-obese men. There may be two possible mechanisms for this. Obese men have increased serum estrogen levels due to enhanced conversion in peripheral fat of testosterone to estradiol and androstenedione to estrone. This may protect against bone loss.[15] Also, excess body weight increases the loading stress to the spine, which stimulates bone formation. In postmenopausal obese women, there is increased conversion of adrenal androgens to estrogens in fat tissue and this leads to decreased bone loss.[13]

Another factor that increases the risk for osteoporosis is the presence of many other diseases. There are many connective tissue diseases related to osteoporosis. Osteogenesis imperfacta is an inherited disease associated with blue sclera, deafness, thin skin, and impaired formation of type I collagen. The onset is during childhood. The disease results in premature spinal osteoporosis. Other common connective tissue diseases also include homocystinuria and autosomal recessive disorder.. These diseases result in an increase in homocysteine and other metabolites that interfere with cross linkage of collagen. This interference with collagen may lead to osteoporotic changes.[13]

There are also many types of endocrine diseases that are associated with osteoporosis. Endogenous or exogenous hyperadrenocorticism decreases bone formation and increases bone resorption. Hyperthyroidism increases bone turnover, however, this is also accompanied with an increase in bone formation.

Gastrointestinal diseases may lead to osteoporosis because of the effects of the impairment in the absorption of calcium and vitamin D. Bone marrow disorders also produce effects of osteoporosis in 10% of patients. There is an increase in local production of lymphotoxin, interleukin-l, and other cytokines by bone marrow cells. Osteoporosis may also occur when there is cancer related to bone marrow because of the above mentioned increases in local production.

There are many types of osteoporosis. Idiopathic Juvenile osteoporosis occurs in boys and girls during the ages of 8-14. There is usually an acute and active phase. The acute phase usually runs 2-4 years, then remits. In the active phase, there is growth arrest and multiple fractures. The etiology of this disease is unknown, but some do believe that hormones may be a factor although there are cases of the disease that occurred in children before puberty (when hormones are most active).

Achondroplasia (dwarfism) is a different disease characterized by a defect in bone and cartilage and is not related to Idiopathic Juvenile osteoporosis.

Idiopathic osteoporosis in young adults occurs in both sexes equally. The cause of this disease is unknown. It has the etiology of several distinct disorders. There are usually multiple vertebral fractures over 5-10 years along with a loss of height of up to 6 inches. The serum values for calcium, phosphorus, and alkaline phosphatase are all found to be normal.. Also, there are both low and high bone turnover rates. The disease characteristics are broad and varied. [13]

There are two types of involutional osteoporosis. Involutional osteoporosis occurs as a result of the aging process. It is characterized by a progressive decline of normal physiological functioning. Type I is postmenopausal and occurs in women 15-20 years after menopause. The rate of trabecular bone loss in these women is 2-4 times greater than that of other postmenopausal women of the same age without fractures. However, the rate of cortical bone loss is only slightly greater.. The following occurs: accelerated bone loss, decreased secretion of parathyroid hormone, and increased secretion of calcitonin. This leads to decreased calcium absorption. There are many new drugs that address these occurrences, they will be discussed later..

Type II osteoporosis occur in both men and women age 70 and over. Women get it two times as much as men. It results from slow bone loss over the course of several decades. The bone density values in parts of the appendicular skeleton fall below normal. This may suggest an even loss in cortical and trabecular bone. Age related factors play an important role in this type of osteoporosis. There is decreased osteoblast function and decreased calcium absorption. The vertebral fractures that occur are the wedge type. These fractures lead to dorsal kyphosis. Trabecular bone thinning is responsible for the painless vertebral deformation. [13]

Bone loss occurs without symptoms, therefore, osteoporosis is not usually discovered until a patient has a fracture. [10] Therefore, the following are common symptoms of the disease: back pain caused by vertebral compression, height loss, spinal deformity(kyphosis with a loss of 4-8 inches in height), fractures of vertebrae(about one per year in the beginning of the disease), hips, wrists, and other bones. [13]

After osteoporosis is suspected, typically bone mineral density tests are done for the following reasons: to confirm the diagnosis, estimate the severity of bone loss, and determine the effectiveness of treatment.

Single energy quantitative computed tomagraphy (QCT) measures bone density by using CT scanners and a calibration phantom. Accuracy is

poor and reproducibility is satisfactory. There is also a high level of radiation exposure. Radiation exposure for a single-energy CT measurement is 100-300 mrem but may go as high as 500-1000 mrem.[12]

A dual-energy QCT is more accurate, however the reproducibility is poorer and there is a higher level of radiation exposure. The radiation exposure is between 5-10 mrem. This is about 1110 of a standard chest x-ray, so the measure can be taken fairly often without excessive radiation exposure. It has the ability to measure exclusively the metabolically active trabecular bone in the center of the vertebral body."[12]

Transmission scanning, dual-photon absorptiometry, allows bone density to be measured independently of soft tissue thickness and measures the entire vertebrae. It emits two photoelectric peaks that allow bone density to be measured independently of soft tissue thickness and composition."[12]

The most recent technique used to measure bone density is the dual-energy x-ray absorbtiometry. It has better reproducibility, shorter scan time, and is capable of measuring the density of the entire skeleton.[13] It uses two x-ray beams with different levels of energy. After eliminating soft tissue absorption, the absorption of each beam by bone is used to calculate the bone mineral density. It is a portable device that is easy to use and convenient.[4]

Bone turnover rates can be measured by assessing the levels of biochemical markers associated with bone resorption and formation that are released into blood or urine. These tests are inexpensive and non-invasive. Some newly recognized markers for bone resorption measured in urine include: pyridinium cross links of collagen, deoxypyridinoune, and pyridinoline. They are highly sensitive and specific for the degradation of bone collagen. Serum markers include bone specific alkaline phosphatase and osteocalcin. They are also sensitive and bone formation specific. Together these tests provide an index of bone turnover rate.[2]

After the disease has been confirmed, the following are the treatment options that are currently available. Many of these treatment options can also be applied to help prevent the disease from occurring in the first place. One of the most common is exercise that keeps muscles strong and toned. The best type of exercise to help keep and maintain healthy, strong bones is weight-bearing activities. They help maintain existing bone by stimulating bone formation. It is also encouraged to incorporate flexibility and balance training in exercise programs. It helps maintain bone mass and reduces the risk of falling by 25%3

One study assessed the effect of back extensor strengthening on osteoporosis. Thirty-six women over the age of 40 were studied. They all

had radiological evidence of either osteopenia (precursor to osteoporosis) or osteoporosis. The study found that those with higher levels of activity (such as back extensor strengthening) had greater bone mineral density than those who had low activity levels (no back extensor strengthening). Therefore physical activity promotes or maintains bone mineral density.[13]

Another study was done to determine whether standing posture exercises could improve thoracic kyphosis and balance in women with osteoporosis. Forty-eight postmenopausal women 59-89 years of age were studied. All of the women were diagnosed with osteoporosis. Control subjects continued their normal activities. Exercise subjects performed five standing posture exercises daily. The study suggests that standing posture exercises may be effective in improving one measure of balance in women diagnosed with osteoporosis. Further studies would be required to establish a long-term effect..[14] Therefore doing standing posture exercises after being diagnosed with osteoporosis could improve a women's thoracic kyphosis and balance.

Another type of exercise treatment is the use of orthotics. A randomized controlled study was done to determine the effect of two different types of back orthoses on back strength in patients with osteoporosis. The study included 45 women between the ages of 43 and 87.

They were randomly assigned to one of three groups. Each group was in the program for a total of 16 weeks. The first group utilized a conventional thoracolumbar support, with the back orthotic used continuously whenever patients were out of bed. The second group was called the Posture Training Group, they wore a weighted kypho-orthosis for four hours twice a day. The third group used no orthotic. All three groups went through body mechanic training, deep breathing training, and seated back extension instruction. Back extensor strength and grip strength were measured before, during, and after training. Patients in the Posture Training Support group showed significant improvement in back extensor strength. The study concluded that the posture training support device increases back extensor strength. This may help prevent compression fractures by providing the weight bearing forces needed to prevent fractures. [7] Therefore, a posture training support device might be a good option for those who have weak back extensors as a result of osteoporosis.

Eating a nutritionally balanced diet while young prevents the onset of adult chronic diseases such as heart-related diseases. Meals should be rich in calcium and vitamin D. This prevention method is also used to treat cases of osteoporosis. A study done on medical nutrition therapy used people with osteoporosis. Medical nutritional therapy included an evaluation of the

patient's health history, social status, and nutrient intake. Based on the assessment, a nutrition care plan was developed and implemented. Nutritional intervention in hip fracture patients showed a significant decrease in mortality and hospital stay time. [11]

Another one of the most common methods of treating osteoporosis is the use of medications. These include Evista, Fosamax, and hormone replacement therapy. However, administration of estrogen is the most common treatment of osteoporosis. Estrogen helps prevent bone loss by acting directly on bone cells to reduce bone resorption. A dose of 0.3mg/day, with calcium supplements has been proven to be as effective as higher doses of estrogen. However, there are some side effects of hormone replacement therapy. Some of these side effects include breast tenderness, mood disturbances, vaginal bleeding, and migraine headaches. Postmenopausal administration of estrogen decreases the occurrence of vertebral and hip fractures by 50%.[13] The exact mechanism of how estrogen prevents bone loss is not known, but there have been some theories: it promotes calcitonin synthesis, it increases vitamin D availability, it inhibits the release of resorptive agents, and it inteferes with bone resorption through bone cell receptors."

Raloxifene has been proven to reduce the risk of vertebral fractures in women with osteoporosis. Raloxifene hydrochloride is a nonsteroidal benzothiophene that binds to estrogen receptors and inhibits bone resorption without stimulating the uterine endometrium in postmenopausal women. **In** fact, 60mg/day of the drug decreased the risk of a vertebral fracture by 30-50%.8 7,705 women between the ages of 31 and 80 participated in this study. All of the women had been post-menopausal for at least two years and had osteoporosis."

Daily oral treatment with 10mg of biophosphonate alendronate sodium (Fasamax) progressively increases the bone mass of the spine, hip and total body in postmenopausal women with osteoporosis according to randomized double blind, placebo-controlled multi-center study. Bisphosphonates are non-hormonal bone specific agents that bind to bone surfaces and inhibit bone resorption. They are the best bone protection agent.. They also work regardless of the patient's age or bone mineral density. [13]

Alendronate prevents vertebral fractures, progression of vertebral deformities and loss of height.. The study included 994 post-menopausal women with osteoporosis, each partcipant took 5 or 10 mg/day for 3 years or 20 mg for 2 years followed by 5mg for the third year.. All the women also

took 500mg/day of calcium. Findings from the study included the following: Bone mineral density increased in the lumbar spine, femoral neck, trochanter, and total body in all the alendronate groups and decreased in the placebo group, the difference in bone mineral density in the spine was 8.8%, 7.8% in the trochanter, and 2.5% in the whole body, and alendronate also cut the incidence of new vertebral fractures in half (3.2 % in the alendronate group, and 6.2% in the control group)."

There was a study done to review the use of alendronate in the postmenopausal women with osteoporosis. The review came to the conclusion that alendronate (along with calcium) is associated with improvement in bone density in hip sites. It is in the hip because this is where there is a greater degree of cortical bone present.. Alendronate is beneficial to those who do not respond well to other treatments such as the use of calcium and vitamin D or estrogen."

Fluoride is another drug used for osteoporosis. It stimulates osteoblastic proliferation and formation of new bone. However, excessive exposure to fluoride may cause abnormal bone formation, micro fractures, and gastric bleeding. "Cyclical, intermittent use of a lower dose of less bioavailable, slow-release sodium fluoride has been shown to maintain serum fluoride at appropriate levels and to stimulate normal bone

formation." This study was a randomized, placebo-controlled study of a slow-release form of sodium fluoride to assess its effect on spinal fracture. Women who had radiological evidence of osteopenia and osteoporosis were randomly selected to receive either fluoride and calcium or a placebo. The treatment group received two cycles of slow-release sodium fluoride, 25 mg twice daily (12 months on therapy, 2 months off) and calcium citrate, 400 mg calcium twice daily. Concurrent treatment with estrogen was similar in both groups (13 of 48 patients in the treatment group and 16 of 51 in the placebo group). In the fluoride group, the L2-L4 bone mineral contented increased substantially by 4-6% per year in all four years of the study. The L2-L4 bone mineral content in the placebo group did not change substantially in any year. Body height decreased significantly more in the placebo group. No significant difference was apparent in minor gastrointestinal and musculoskeletal side effects. The conclusion was that the delayed release of fluoride along with intermittent withdrawal of fluoride and the provision of calcium citrate is a safe treatment and has a positive response on bone formation. The short-term withdrawal of fluoride has been shown to overcome the attenuated osteoblastic activity that occurs with continuous fluoride therapy.[1] Therefore, fluoride used intermittently along with calcium citrate may be used to treat osteoporosis.

In conclusion, osteoporosis is a complex pathological process that occurs in bone. There are many diseases that increase the likelihood of getting osteoporosis. There are also many lifestyle factors that increase the likelihood. Diagnosis of the disease can be done using different methods, CT scans being one of the most common. After diagnosis has been confirmed, there are many treatment options available including hormone replacement therapy. Overall, osteoporosis can be a devastating disease, but there are many ways that its effects can be diminished, the most important being the individual..

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