



A REVIEW ON NUTRITIONAL MANAGEMENT IN OSTEOPOROSIS

**Nitin E. Patil¹, Dr. Kantilal B. Narkhede², Dr. Anuradha P. Prajapati³,
Dr. Sachin B. Narkhede⁴, Dr. Shailesh Luhar⁵, Oza Vrushant⁶**

1,2,3,4,5,6 Smt B.N.B Swmainarayan Pharmacy College

Article DOI: <https://doi.org/10.36713/epra15263>

DOI No: 10.36713/epra15263

ABSTRACT

Osteoporosis is condition of bone being porous because of various factors such as lack of nutrition, age, some therapeutic remedies like steroids, disease condition such as hypertension, hyperthyroidism, and diabetes mellites. There are various allopathic treatment remedies to treat the osteoporotic patients such as Bisphosphonates, Calcitonin. But since these all are allopathic remedies they have some short of side effects with them. So, use of nutrition base product which are natural and have no or negligible side effects. These products are calcium, vit D & k, Essential fatty acids, dietary protein, and dairy products. Use of these product can improve the bone health from the osteoporotic condition.

INTRODUCTION

Osteoporosis, literally a condition of porous bones, affects 10 million people a year in the United States and 200million people worldwide. In addition, 18 million people have low bone mass (osteopenia), which puts them at risk for osteoporosis. The basic problem is that bone resorption (breakdown) outpaces bone deposition (formation). In large part this is due to decrease or depletion of calcium from the body—more calcium is lost in urine, faces, and sweat than is absorbed from the diet. Bone mass becomes so depleted that bones fracture, often spontaneously, under the mechanical stresses of everyday living. For example, a hip fracture might result from simply sitting down too quickly. Osteoporosis afflicts the entire skeletal system. In addition to fractures, osteoporosis causes shrinkage of vertebrae, height loss, hunched backs, and bone pain. Osteoporosis primarily affects mid-aged and elderly people, 80% of them women. Older women suffer from osteoporosis more often than men for two reasons: (1) Women's bones are less massive than men's bones, and (2) production of oestrogens in women declines dramatically at menopause, while production of the main androgen, testosterone, in older men wanes gradually and only slightly. Oestrogens and testosterone stimulate osteoblast activity and synthesis of bone matrix. Besides gender, risk factors for developing osteoporosis include a family history of the disease, thin or small body build, an inactive lifestyle, cigarette smoking, a diet low with calcium and vitamin D, more than two alcoholic drinks a day, and the use of certain medications. Although the bone is adequately mineralised, it is fragile and microscopically abnormal, with loss of internal structure. Peak bone mass occurs around 35 years and then gradually declines in both sexes. Low oestrogen levels after the menopause are associated with a period of accelerated bone loss in women. Thereafter bone density in women is less than in men for any given age. A range of environmental factors and diseases are also associated with decreased bone mass and are implicated in development of osteoporosis. Some can be influenced by changes in lifestyle. Exercise and calcium intake during childhood and adolescence are thought to be important in determining eventual bone mass of an individual, and therefore the risk of osteoporosis in later life. As bone mass decreases, susceptibility to fractures increases. Immobility causes reversible osteoporosis, the extent of which corresponds to the length and degree of immobility. For instance, during prolonged periods of unconsciousness, osteoporotic changes are uniform throughout the skeleton, but immobilisation of a particular joint following fracture leads to local osteoporotic changes in involved bones only. Osteoporosis is diagnosed by taking a family history and undergoing a bone mineral density (BMD) test. Performed like x-rays, BMD tests measure bone density. They can also be used to confirm a diagnosis of osteoporosis, determine the rate of bone loss, and monitor the effects of treatment. There is also a relatively new tool called FRAX[®] that incorporates risk factors besides bone mineral density to accurately estimate fracture risk. Patients fill out an online survey of risk factors such as age, gender, height, weight, ethnicity, prior fracture history, parental history of hip fracture, use of glucocorticoids (for example, cortisone), smoking, alcohol intake, and rheumatoid arthritis. Using the data, FRAX[®] provides an estimate of the probability that a person will suffer a fracture of the hip or other major bone in the spine, shoulder, or forearm due to osteoporosis within ten years. ^[16-17]

In 2015, direct medical costs totalled \$637.5 million for fatal fall injuries and \$31.3 billion for nonfatal fall injuries. During the same year, hospitalizations cost an average of \$30,550 per fall admission, totalling \$17.8 billion. By 2025, the cost of fractures in



the United States is expected to exceed \$25 billion each year to treat more than three million predicted fractures. Management of osteoporosis and its associated consequences is necessary to improve quality of life and reduce economic burden on the health care system. It will also help to decrease medical visits, hospitalizations, and nursing home admission.^[1]

In recent years, major therapeutic advances in osteoporosis treatment have been made as scientists gain a greater understanding of bone morphology and the underlying mechanisms causing osteoporosis. This article will review the pathophysiology, etiology, screening, and diagnosis of osteoporosis; selected professional guidelines and pharmacological management; pharmacological options, and the cost-effectiveness of those options.^[1]

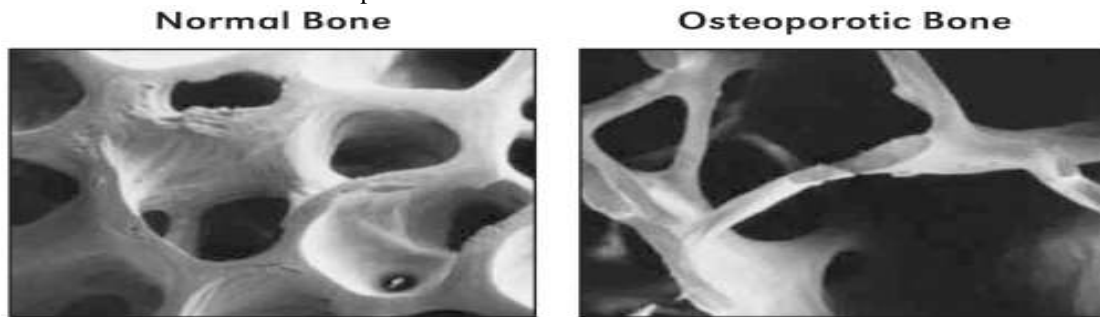


Fig.1 Comparison Between Normal and Osteoporotic Bone [19]

CAUSES AND RISK FACTORE

Nutritional deficiency ^[2]

Health-promoting behaviours, such as consuming a healthy diet, can reduce the impact of chronic diseases such as osteoporosis and cardiovascular diseases. A dietary pattern with high intake of dairy products, fruits and whole grains may contribute positively to bone health, and a study on Chinese older women in Hong Kong showed that higher "vegetables–fruits" and "snacks–drinks–milk products" pattern scores were associated with reduced risk of cognitive impairment. Consumption of nutrient such as calcium, vit D and K helps to improve bone health.

Genetic Factor ^[2]

The genetics of osteoporosis, a key focus in bone biology research, reveals that bone mineral density (BMD) variation is largely influenced by genetic factors. The vitamin D receptor (VDR) gene, specifically the BsmI polymorphism (genotype bb), has a significant impact on BMD, particularly in the lumbar spine. This variant is associated with higher calcium absorption and increased BMD levels, affecting bone metabolism through various mechanisms.

Medication ^[2]

Synthetic glucocorticoids are administered to treat disorders caused by autoimmune, pulmonary, and gastrointestinal diseases, as well as in patients receiving organ transplantation and with malignancies.

Hyperthyroidism ^[2]

Primary hyperparathyroidism (PHPT) is a calcium metabolic disorder with the highest incidence in postmenopausal women. Several studies have shown decreased BMD in patients with PHPT.

Smoking ^[2]

Smoking is a risk factor for osteoporosis and is associated with decreased bone mass and an increased risk of fractures.

Hypertension ^[3]

Studies have shown that high blood pressure is associated with abnormalities of calcium metabolism. The mechanism by which this occurs is due to a defect in the kidney's ability to handle calcium.

Diabetes ^[3-4]

Postmenopausal women who have diabetes or in whom Diabetes develops are at higher risk for hip fracture than Unaffected postmenopausal women.



COMPLICATRION

Fragility Fracture ^[5]

A fragility fracture is defined as a fracture that occurs because of a low-energy force that is insufficient to break normal bone. The most common locations for these fractures are the spine, hip, pelvis, proximal humerus, forearm, and wrist. Current estimates state that for the average Caucasian woman older than 50, the lifetime risk of a fragility fracture ranges from 33% to 45%. The incidence of these fractures increases dramatically in persons older than 65.

Hip Fracture ^[5]

Hip fractures remain the most serious fragility fractures in terms of morbidity and mortality; about half of these individuals never regain their previous functional capacity. One-year mortality rates are estimated to be in the range of 14% to 36%. The nature of the fall must be determined, as these patients often have multiple medical comorbidities; possible causes of the fall, such as stroke, myocardial infarction, or dehydration, must be delineated because circumstances have an impact on medical optimization for surgery.

Colle's Fracture ^[6]

During the acute period after a Colle's fracture, substantial pain, and restriction in function OCCU. Often, potentially long-term functional consequences can be minimized by physical therapy and exercise; unfortunately, some individuals with wrist fractures experience restricted activity, chronic pain, and loss of function. This may be especially true in patients in whom algodystrophy develops, also known as reflex sympathetic dystrophy, which requires immediate initiation of treatment.", 2o in most patients, however, quality of life changes resulting from distal forearm fractures are not overwhelming.

Pain ^[6-7-8]

Pain from osteoporosis challenges the patient's coping skills, often in a way that causes a decline in physical and psychological well-being. Two types of osteoporotic pain exist: (1) acute pain that accompanies a fracture and (2) chronic pain that develops over time. Each type of pain creates a unique predicament for the individual with osteoporosis as well as for the health care professional handling the case.

Psychological Disfunction ^[6]

The physical deformity and functional limitations associated with osteoporosis are evident in individuals with established disease. The psychological impact, although not visible, can be as serious and debilitating.

PATHOPHYSIOLOGY

Compromised bone strength predisposing a person to an increased risk of fracture is defined as osteoporosis. Bones provide structure for the body, protection for the organs, and storage for minerals, such as calcium and phosphorus, that are essential for bone development and stability.

Skeletal formation begins during the sixth week of embryonic development. The skeletal mass of infants doubles in the first year of life, and 37% of the total skeletal mass is accumulated during adolescence. Skeletal growth continues until genetic height is attained, but bone mineralization continues until the third decade. Calcium, vitamin D and weight bearing activity are the key components of bone growth. Other factors, including growth hormone, are involved in optimal bone production, but their roles have not been fully elucidated.

Factors which have a role in regulating bone function are hormones and growth. Oestrogen and testosterone have inhibitory effect of bone breakdown on bone remodelling. Cytokines which influence remodelling are identified, such as receptor activator of the nuclear factor kappa-B ligand (RANKL) osteoprotegerin (OPG) of tumour necrosis factor (TNF) receptor and RANKL is produced by osteoblasts that bind to RANK receptors on osteoclasts, leading to the activation and maturation of osteoclasts and culminating in bone resorption. The production of OPG by osteoblast cells counterbalances maturation of osteoclasts and their activity by acting as a decoy receptor of RANKL. Thus, a relative concentration of OPG and RANKL in bone determines its strength and mass. Recent advances in molecular bone biology have identified a potent protease cathepsin K (CatK) which is secreted by activated osteoclasts during bone resorption. Resulting in degradation of bone matrix and breakdown of mineral components of the bone tissue. Parathyroid hormone plays an important role in bone formation by indirectly increasing the proliferation of osteoblasts through regulation of calcium homeostasis. ^[9-12]

ALLLOPATHYC REMIDIES ^[18]

Two types of agents are currently used for treatment of osteoporosis:

1. Antiresorptive drugs that decrease bone loss.
 - a. Bisphosphonates



- b. Calcitonin
 - c. selective oestrogen receptor modulators (SERMs)
 - d. calcium.
2. Anabolic agents that increase bone formation
 - a. PTH
 - b. teriparatide.

NEUTRITIONAL MANAGEMENT

Nutritional management in osteoporosis required certain nutrients that full fill the need of body to maintain the bone density. It includes certain conventional nutrients with regular intact of that n day to day life and certain supplement that maintain the bone health.

Vitamin D ^[13-14]

Vitamin D plays an essential role in maintaining bone strength and muscle function. This nutrient/cofactor is involved in the intestinal absorption of calcium and phosphorus for bone mineralization and the maintenance of muscle mass as well as various beneficial effects on other organ systems.

Vitamin D is synthesized in the skin in response to sun exposure and taken as part of a balanced diet. Older people synthesize lower levels of vitamin D in their skin (they also tend to have less exposed skin than younger people) and their diets are often low in nutrients. Many older people suffer from vitamin D deficiency.

CALCIUM ^[15]

Peak bone mass is usually reached around age 30; therefore, physical activity and recommended doses of calcium and vitamin D during adolescence and early adulthood will ensure peak bone mass. Calcium is an essential element for the human body and is necessary for many cellular functions. Calcium is not only important for bone health, but it is also essential for neuromuscular activity, blood clotting and normal heart function.

Dietary Protein Intake ^[15]

Malnutrition and malnutrition are common in older people and can lead to deficiencies in essential nutrients. Malnutrition, especially protein-energy malnutrition in many older people, is a major risk factor for sarcopenia and frailty. In a small prospective study of Australian hip fracture patients (72% female), 58% of patients admitted were malnourished and 55% had vitamin D deficiency. Raynaud-Simon and colleagues found estimated the prevalence of protein-energy malnutrition to be 4-10% in older people living at home, 15-38% in care facilities, and 30-70% in older hospitalized patients. Questionnaires such as the Mini Nutritional Assessment (MNA) or SNAQ65+ have been validated in the elderly and are useful for assessing nutritional status in this regard.

Essential Fatty Acids (EFAs) ^[14]

Recent research confirms that adequate and balanced levels of AGEs in the diet have a positive effect on bone health and that a deficiency in AGEs can be a major cause of osteoporosis. Although the complex mechanisms of bone physiology are not fully understood, many molecular factors integral to bone formation and resorption are controlled or influenced by the availability and action of AGEs. The discovery that animals deficient in essential fatty acids develop severe osteoporosis has prompted new research into the role of these essential nutrients in bone health.

Vitamin k ^[14]

VTK is a fat-soluble molecule that occurs naturally in two forms: (a) VTK-1 (phylloquinone) is synthesized by plants and is abundant in green leafy vegetables and some vegetable oils (soy, canola, and olive); (b) VTK-2 (menadione) collectively refers to a group of compounds commonly synthesized by bacteria that normally colonize the colon, as well as the intestinal metabolism of VTK-1. The "K" component of VTK is derived from the German word for "coagulation" because this essential molecule is an essential coenzyme involved in blood clotting, but recent research has also recognized VTK as a cofactor in many cell-associated biochemical pathways. Growth, brain development 106 and bone health.

CONCLUSION

In osteoporotic condition use of the nutritional supplements like calcium, vit D & K, essential fatty acid, dietary protein, and other supplement has significant improvement in the bone health. These nutrients directly have role in the bone formation and reduce the depletion of bone by various in vivo mechanisms. So, these nutrients play an important role in the bone formation which is required in the osteoporotic condition.



REFERENCE

1. Tu, K. N., Lie, J. D., Wan, C. K. V., Cameron, M., Austel, A. G., Nguyen, J. K., Van, K., & Hyun, D. (2018). Osteoporosis: A review of treatment options. *Pharmacy and Therapeutics*, 43*(2), 92-93.
2. Poursmaeili, F., Kamalidehghan, B., Kamarehei, M., & Goh, Y. M. (2018). A comprehensive overview on osteoporosis and its risk factors. *Therapeutics and Clinical Risk Management*, 14*, 2029-2049.
3. Alagiakrishnan, K., Jubay, A., Hanley, D., Tymchak, W., & Sclater, A. (2003). Role of vascular factors in osteoporosis. *Journal of Gerontology: Medical Sciences*, 58A*(4), 362-366.
4. Nguyen, T. V., Eisman, J. A., Kelly, P. J., & Sambrook, P. N. (1996). Risk factors for osteoporotic fractures in elderly men. *American Journal of Epidemiology*, 144*(3), 255-262.
5. Varacallo, M. A. (2014). Osteoporosis and its complications. *Medicine Clinics of North America*, 98*, 817-831.
6. Gold, D. T., Shipp, K. M., & Lyles, K. W. (1998). Managing patients with complications of osteoporosis. *Endocrinology and Metabolism Clinics of North America*, 27*(2), 485-494.
7. Griffith, J. F., & Genant, H. K. (2012). New advances in imaging osteoporosis and its complications. *Endocrine*, 42*(1), 39-51.
8. Eginster, J. Y., & Burlet, N. (2006). Osteoporosis: A still increasing prevalence. *Bone*, 38*(2 Suppl 1), S4-S9. doi: 10.1016/j.bone.2005.11.024
9. Ferrari, S., Bianchi, M. L., Eisman, J. A., Foldes, A. J., Adami, S., Wahl, D. A., & Stepan, J. J. (2012). Osteoporosis in young adults: Pathophysiology, diagnosis, and management. *Osteoporosis International*, 23*, 2735-2748.
10. Awasthi, H., Mani, D., Singh, D., & Gupta, A. (2018). The underlying pathophysiology and therapeutic approaches for osteoporosis. *Medical Research Reviews*, 1-34.
11. Alexandre, C., & Vico, L. (2011). Pathophysiology of bone loss in disuse osteoporosis. *Joint Bone Spine*, 78*, 572-576.
12. Föger-Samwald, U., Dovjak, P., Semrad, U. A., Schindl, K. K., & Pietschmann, P. (2020). Osteoporosis: Pathophysiology and therapeutic options. *EXCLI Journal*, 19*, 1017-1037.
13. Rizzoli, R., Branco, J., Brandi, M. L., Boonen, S., Bruyère, O., Cacoub, P., & Cooper, C. (2014). Management of osteoporosis of the oldest old. *Osteoporosis International*, 25*, 2507-2529.
14. Genuis, S. J., & Schwalfenberg, G. K. (2007). Picking a bone with contemporary osteoporosis management: Nutrient strategies to enhance skeletal integrity. *Clinical Nutrition*, 26*, 193-207.
15. Sunyecz, J. S. (2008). The use of calcium and vitamin D in the management of osteoporosis. *Therapeutics and Clinical Risk Management*, 4*(4), 827-836.
16. Tortora Gerard j, Derrickson Bryan, principles of anatomy and physiology, 14th edition WILEY publication, page no.188
17. Waugh A, Grant A, Ross & Wilson Anatomy and physiology in health and illness pub, 11th edition, page no. 431
18. Rang HP, Ritter JM, Flower RJ, Henderson G, Rang & Dale's pharmacology, Elsevier publication, 6th edition, page no. 473-440.
19. www.ncbi.nlm.nih.gov