

Egyptian Guidelines
for
the Diagnosis and Management
of
OSTEOPOROSIS

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Preface

Osteoporosis has emerged as health care problem for the last 20 years for 3 important factors:

1. The prolonged life expectancy for individuals due to increased health services.
2. New diagnostic tools have emerged which made Osteoporosis easily and early diagnosed.
3. New armamentarium of drugs still growing for early treatment and prevention of fractures.

Although these advances have been taking place, Osteoporosis is still under diagnosed. This disease is silent but unfortunately pain occurs when fractures take place. This has a significant financial and socio economic impact on the patient and his family.

This disease is preventable and once prevented can lower the incidence of fractures. Even if the first fracture occurs, proper treatment can prevent occurrence of a second fracture.

A working group of key opinion leaders in the field of Osteoporosis management constituted the editorial board of these Guidelines supported by Servier, with its growing special interest in this field, took the initiative to put the first national Guidelines that was endorsed by the board of the Egyptian Osteoporosis Society based on a systematic review and a critical appraisal of the currently available literature.

The Guidelines will be reviewed and updated every 2 years in order to go hand in hand with the international Guidelines of different societies.

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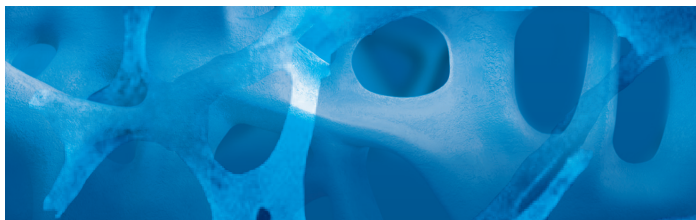
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Background

What is Osteoporosis?

In 1842, an English Surgeon named “Astley COOPER” observed that the bones of old people become thin in their shell and spacious in their texture. In 1940, “Albright” defined the crucial connection between menopause and Osteoporosis in women.

In 1958, a Canadian pathologist “William BOYD” wrote: “Postmenopausal Osteoporosis is unfortunately a very common condition accounting for the increased frequency of fractures, caused by a relatively slight trauma in elderly women”. In 1983, “Riggs and Melton” clearly recognized and distinguished postmenopausal and Senile Osteoporosis.

The steadily increasing number of publications and scientific conferences devoted to Osteoporosis is a strong evidence of the interest aroused by this disease.

There appears to be two main reasons for this phenomenon:

1. The medical profession and all the influential people concerned with public health have become aware of the socio-economic burden imposed by this disease.
2. In addition, the recent advances made in our knowledge and the understanding of bone metabolism, have given us a greater insight into Osteoporosis.

Osteoporosis is neither a medical curiosity, nor a benign disease. It is a malady whose incidence rises with age. Consequently, the steady increase in life expectancy and the corollary aging of population make Osteoporosis a real public health problem. The prevalence of Osteoporosis is not precisely known as many texts suggest. The difficulty lies in the precise diagnosis of the disease.

As the number of people in the elderly population worldwide increases, the number of patients who can be expected to suffer from Osteoporosis will consequently increase.

In the United States of America, it is estimated that by the year 2025, there will be 60 million Americans over the age of 65 years, with the consequent increase in the number of elderly people suffering from Osteoporosis, as well as increasing incidence of osteoporotic fractures.

In these terms alone, it can be readily appreciated that Osteoporosis is a public health problem that is going to become increasingly more important.

It is not only more common than most of other serious diseases, but also much more insidious in character. Like a silent thief in the night, it weakens the bones by slipping away with their substance. Its victims remain wholly unaware that they are losing bone little by little, for years



to come and with no end. These bones though not diseased become weak and brittle, and produce no symptoms until one suddenly breaks. Only then, Osteoporosis is recognized and diagnosed because a fracture has occurred. The underlying bone loss which weakened the broken bone, together with other parts of the skeleton, is more likely to be overlooked than investigated and treated. Once the fracture has healed, Osteoporosis may go unrecognized once again until another fracture occurs which may be fatal or disabling. Sadly, this is still the natural history of Osteoporosis in too many postmenopausal women and old people of both sexes.

A story of lost opportunities, it is hard to believe that less than 15 years ago, we were only beginning to develop instruments to measure bone mineral density. Today, Osteoporosis, with its resultant fractures and physical disabilities, is recognized as a major public health problem. In addition, tremendous development of devices that can non-invasively measure bone mass has occurred. Along with the increased diagnostic capability, a number of bone active agents have been developed and made available to prevent and treat this disabling disease. In addition, we also understand how treatments, such as Calcium and exercise, are essential for bone health.

Interaction of genetic and nongenetic factors have a great influence on peak bone mass: Multiple factors contribute to Osteoporosis, including nongenetic (e.g.: environment and nutrition), and genetic factors. Achievement of peak bone mass, which is mainly determined by alterations in bone size and volumetric density, is a critical determinant of the risk of Osteoporosis. Complex and selective genetic, hormonal, nutritional and other environmental factors, all interact closely to control these developmental processes. A variety of mechanisms, and multiple influences on bone homeostasis govern skeletal growth, therefore, genetic control of bone mass implicates numerous genes of variable importance during an individual's lifespan.

I. Egyptian Guidelines for the Diagnosis of Osteoporosis

I. Egyptian Guidelines for the Diagnosis of Osteoporosis:

- 1.1 The need for Guidelines**
- 1.2 Objectives of Guidelines**
- 1.3 Definitions**
- 1.4 Risk factors**
- 1.5 Diagnosis**

1.1. The need for Guidelines:

Osteoporosis is a major health problem in most countries including Egypt, and its prevalence is increasing. The public health and clinical importance of Osteoporosis lies in the fractures risk associated with the disease and its economic and social impact.

Although osteoporotic fractures are an important cause of morbidity, disability and mortality, they are preventable. The International Osteoporosis Foundation (IOF) recommended the establishment of nation-specific Guidelines and requested to take into consideration the specificities of each and every care environment. With this in mind, an Advisory Board for Consultancy (ABC) including the different medical specialties related to the subject of Osteoporosis, set itself

the task of establishing Guidelines for the diagnosis and management of Osteoporosis in Egypt. These Guidelines are based on the evidence-based data in the published literature and took into consideration the specificities of the medical care and services in Egypt.

The need for Guidelines is extremely important for Egypt:

Firstly, there is a wide range of diagnostic and monitoring tools available. We need to identify those at risk of, or suffering from, Osteoporosis, and it is important to identify the most effective and economic tool of those available.

Secondly, across Egypt, there is a significant variation in the availability of physicians interested in Osteoporosis, in the availability of diagnostic tools and in the referral and treatment rates. This requires Guidelines to establish priorities in those who will be treated and the tool to be used.

Thirdly, Guidelines explore the selection of patients for referral to more specialized centers for further investigations and monitoring. The objective of the Guidelines is to ensure the timely identification of those individuals at highest risk of Osteoporosis, as well as those who already have the disease.

Fourthly, Guidelines pay particular attention to treatment options that can be used in those patients to reduce their increased risk of fracture.

1.2. Objectives of Guidelines for early diagnosis:

The Guidelines shall optimize the use of the existing tools in early diagnosis of Osteoporosis as it will help the General Practitioner as well as the specialist in screening persons with or without risk factors **to avoid the occurrence of fractures especially the first fracture.**

1.3. Osteoporosis definitions:

1. WHO definition:*

Osteoporosis is a skeletal disorder characterized by a low bone mass and microarchitectural deterioration of bone tissue with a consequent increase in bone fragility and susceptibility to fracture.

Normal BMD **	T-score no more than -1 SD below the young adult mean.
Osteopenia	T-score between -1.0 and -2.5 SD.***
Osteoporosis	T-score equal to or less than -2.5 SD.
Severe or Established ****	T-score below -2.5 SD and fragility fracture.
Osteoporosis	

Table 01

* WHO: World Health Organisation.

** BMD: Bone Mineral Density.

*** SD: Standard Deviation.

**** Occurrence of fragility fracture denotes severe Osteoporosis regardless of T-score (Editorial board).

II. NIH* definition: (New definition):

Osteoporosis is a skeletal disorder characterized by compromised bone strength predisposing a person to an increased risk of fracture.

(Bone strength primarily reflects the integration of bone density and bone quality).

**NIH: National Institute of Health.*

Risk Factors for Osteoporosis:

- Postmenopausal women.
- Age.
- Family History of Osteoporosis and / or Osteoporotic fractures.
- Falls.
- Inadequate intake of Calcium and Vitamin D.
- Clinical disorders associated with Osteoporosis.
- Life style.
- Existing fragility fracture.
- Drugs inducing Osteoporosis.

Points to remember:

- Estrogen is a main determinant for attaining the peak bone mass, more bone formation occurs with optimum estrogen levels.
- The drop in the Bone Mineral Density (BMD) is considerable in the first five years after menopause especially in the first year.

- The most important time to guard the bone of a woman against Osteoporosis is the first few years after menopause.

1.4. Risk factors for Osteoporosis:

1.4.1. Postmenopausal women:

1.4.1.1 Menopause:

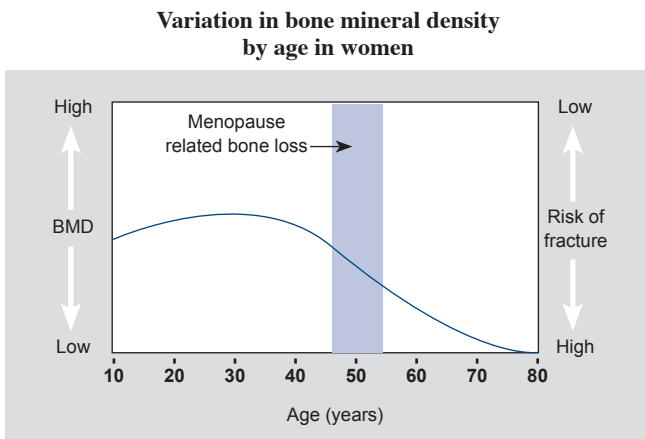


Figure 01

By definition, menopause is: “Cessation of menstruation for six consecutive cycles”.

The period of time needed for the transition from the reproductive life to the senile life is called the “climacteric”, it usually takes about ten years. During this gradual transition to senile life, there is an acute event that is very evident, which is the cessation of menstruation or menopause.

Menopause usually occurs around the age of fifty, due to depletion of the ovary from the ova that traveled to the ovarian cortex through the root of the mesentery during the intrauterine life, these ova secrete estrogen from the granulosa cells that developed after puberty under the effect of FSH (Follicle Stimulating Hormone) secreted from the anterior pituitary. So unlike men, the ova in the female are all formed during the intrauterine life and no more ova could be formed later, women are using ova from a constant stock that will be depleted after a certain time and could not be refilled or re-supplied again.

1.4.1.2 Estrogen and bone:

Estrogen receptors are available in all body tissues. Estrogen is responsible for female body changes starting from puberty and lasting till its decline at menopause. One of the most important functions is the formation of the peak bone mass.



Estrogen helps in the homeostasis of Calcium in bone, also it is essential for the bone matrix and collagen fibers all over the female body.

Estrogen activates the osteoblasts and give it the upper hand over the osteoclastic activity, hence more bone formation occurs with optimum estrogen levels.

Estrogen deficiency:

Decline of estrogen level, makes woman suffer as the estrogen level becomes lower than that her body has adjusted itself to.

The female starts to suffer from hot flushes, headache, easy fatigability, depression and many other symptoms that differ from one to another.

Bone is very vulnerable for estrogen decline. The drop in the Bone Mineral Density (BMD) is considerable in the first five years after menopause especially in the first year. Five years after menopause, although estrogen level is still low or even lower, the decline in BMD is as the comparable age groups. The most important time to guard the bone of a woman against Osteoporosis is the first few years after menopause.

If, for a reason or another, we have to induce iatrogenic menopause whether surgical or medical, we must consider seriously the deleterious effects of sudden estrogen

deprivation on BMD. Also, if we are supporting the female body with exogenous estrogen, we must care about the negative impact of withdrawing this support on the BMD.

Postmenopausal Osteoporosis endanger the physical capability of the woman, who is usually the back bone of a family. A 50 years old woman is still in the most fruitful years of her life, unlike senile Osteoporosis that affect human beings at the age of 70.

1.4.1.3 Premature menopause:

All causes of gonadal failure at young age cause loss of skeletal mass, this means that the presence of gonadal hormones is clearly important in maintaining bone mass. Examples of premature menopause include anorexia nervosa, exercise-induced gonadal failure (Professional Athletes) and prolactinaemia where this induces changes in gonadal hormones. All the previously mentioned examples will finally affect the efforts to attain a peak bone mass and consequently Osteoporosis will occur in very young females secondary to ovarian dysfunction.

1.4.1.4 Andropause:

By the time men are between the ages of 40 and 55, they can experience a phenomenon similar to the female menopause,



called andropause. Unlike women, men do not have a clear-cut sign such as the cessation of menstruation to mark this transition. Both however are distinguished by a drop in the hormone levels (Estrogen in the female and testosterone in the male). The body changes occur very gradually in men and may be accompanied by changes in the attitude and mood, fatigue, loss of energy and sex drive.

Unlike women, the transition from active life to senile life may take several decades and could be hardly noticed.

Although with age, a decline in testosterone levels will occur in some men virtually, there is no way of predicting who will experience andropausal symptoms of sufficient severity to seek medical help. Neither it is predictable at what age symptoms will occur in a particular individual. Each man's symptoms will be also different.

Studies show that this decline in testosterone can actually put one at risk of other health problems like heart diseases and weak bones. It is often difficult to realize that the changes occurring are due to testosterone drop or other co-morbid conditions, e.g.: hypercholesterolemia.

Starting around the age of 30, testosterone levels drop by about 10 % every decade. At the same time, the increased Sex Hormone Binding Globulin level (SHBG) decreases the level of biologically active free testosterone. It is estimated

that by the age of 50 years, 30% of men will have low testosterone levels that may cause symptoms like low sex drive, psychological changes, decreased muscle mass, loss of muscle strength, increased upper and central body fat, cardiovascular risk and weak bones, it is also estimated that between the ages of 40 and 70 years, the male BMD falls by 15%.

1.4.2. Age:

1.4.2.1 Aging process:

It is a well-known fact that age is a major contributor to the occurrence of Osteoporosis and consequently to osteoporotic fractures. The 10-year probability of experiencing a fracture of the forearm, humerus, spine or hip increases as much as 8-folds between the age of 45 and 85 for women and 5-folds for men.

The cumulative lifetime fracture risk for a 50-year woman with Osteoporosis is as high as 60.

A 55 years old person with a low BMD is at significantly less risk than a 75 years old with the same low BMD. Osteoporotic fractures occur most commonly in men and women over 65 years of age, therefore, it seems prudent to begin the identification of people at high risk for Osteoporosis in their 50s.

Many factors contribute to bone loss in old age: Low protein intake, low Calcium intake, and malnutrition in general have been associated with significant bone loss, at both femoral and spine sites and increases the risk of femoral fractures.

**Incidence of osteoporotic fractures
in women by age**

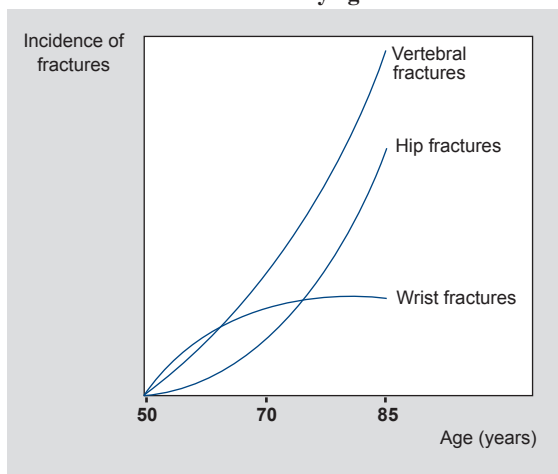


Figure 02

The incidence of all fractures tends to start to rise at, or around, the menopause. The risk of hip fracture increases

markedly after the age of approximately 70 years, owing, in part, to the increased incidence of falls in this older age group, and the increased risk that an older person who falls will land on their hip. Conversely, the risk of wrist fracture tends to decrease with age, possibly because an older person has less likelihood of falling on to their outstretched arm. The incidence of osteoporotic vertebral fractures, which are largely unrelated to trauma, increases at a more or less steady rate after the menopause.

1.4.2.2. Age-related bone loss:

Bone trabecular changes with age:

The Arrangement of bone trabeculae may change with the following:

- Age.
- Change of activity, e.g.: sudden drop in activity due to retirement.
- Load applied on it (stresses).
- Co morbid diseases.
- Weight changes (body mass index).

The changes involve: diameter of bone fibers, size of pores, breaking of cross links, buckling of bone trabeculae caused by stress applied, this will end up with **fragility fractures**.



Cellular activity: normally, there is a balance and coupling between osteoblast and osteoclast functions through the normal bone cycle.

The bone remodeling cycle:

Osteoblast is the bone forming cell originating from undifferentiated mesenchymal cells, its life span varies from 6 to 12 weeks depending on estrogen level. Its number and activity directly correlates with the estrogen level while **Osteoclast** is a bone erosive cell originating from the macrophage lineage, it is multinucleated with ruffled border. Its life span is 16 weeks. Its number and activity inversely correlates with estrogen level and vice versa.

The mechanical integrity of the skeleton is maintained by the process of bone remodeling, which occurs throughout life. This process of regeneration, degradation, and repair, allows damaged bone to be replaced by new bone. Remodeling can be divided into four phases: resorption, reversal, formation, and quiescence. At any one time, approximately 10% of bone surfaces in the adult skeleton are undergoing active remodeling, whereas the remaining 90% is quiescent. The duration of the remodeling cycle is approximately six months, with the resorption phase taking 10 to 14 days, and formation approximately 150 days.

The bone remodeling cycle

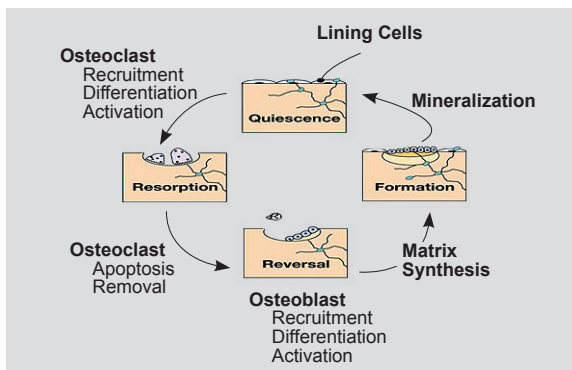


Figure 03

1. Age related changes in osteoblasts occur:

- Decreased osteoblasts number after menopause in women and above the age of 65 in men.
- Shorter life span of cells.
- Decreased cell activity.
- Decreased rate of formation.
- Decreased rate of differentiation.

2. Age related changes in osteoclasts occur:

- Increased osteoclasts number.
- Increased life span.
- Ruffle border is enlarged.
- Increased coalescence.
- Increased activity.

- There is coupling between osteoclastic and osteoblastic activities. Normally, osteoblasts are taking the lead. In Osteoporosis, osteoclasts take the lead.
- Remodeling cycle affected by bone cells goes in favor of resorption, the cycle becomes longer, starts by erosion, and takes six months before the bone can be repaired by osteoblasts. In contrast, in the normal remodeling cycle, the bone resorption phase takes 2 weeks only.

3. Age related changes in mineralization occur bone becomes less mineralized due to:

- Dietary deficiency.
- Loss of appetite.
- Psychological situations.
- Use of diuretics.
- GIT dysfunction: malabsorption diseases, increase in the PH of stomach and in the intestinal motility decrease.
- Negative impact of aging process on protein and collagen synthesis.
- Defects of Vitamin D activation due to loss of subcutaneous fat as well as renal and hepatic dysfunction.

Aging affects the ratio between cortical and medullary cross sectional area of the diaphysis from childhood to senility. This ratio of medulla to cortex increases from 1:2 to 2:1. While the total cross section increases, the BMD may not vary but the bending and axial load to failure is improved, thus with advancing age, the cortical bone quality plays a paramount role.

Effect of Geometry on Long Bone Strength

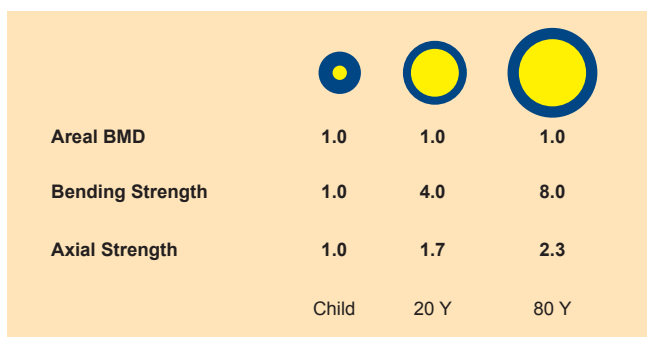


Figure 04

1.4.3. Family history of Osteoporosis and/or osteoporotic fractures:

Heredity plays a major role in the predisposition to Osteoporosis. In youth, 50% of the variance of bone density may be due to genetic factors. The genetic component of peak



bone mass appears to vary between skeletal sites, it is more marked at the lumbar spine than at the forearm, hip or calcis.

Bone mass is also lower in daughters of osteoporotic patients. Maternal genetic factors are thought to contribute to the variations in bone mass of daughters of women with fractures, however it is less obvious after the menopause.

Recent studies have shown that up to 50% of the apparent hereditary or about 30% of the variation of the bone mass may be associated with allelic variations in the vitamin D receptor gene.

The positive history of osteoporotic fractures in the family increases the chances of developing fractures of offsprings of the same family.

1.4.4. Falls:

- It is crucial to evaluate the patients risk of falling especially among old people.
- Fractures are usually associated with falls, a history of factors that increase the risk of falling should be included in the risk assessment.
- Risk factors for falling include those associated with general frailty, such as reduced muscle strength, e.g.: inability to rise from a chair without assistance, impaired balance and low body mass, slower gate.

- Visual impairment is an important risk factor for falls and cataract is the most common cause of visual impairment in the elderly.
- Neurological conditions such as Parkinson's disease should be treated early to reduce the chances of a fall.
- Postural hypotension in elderly hypertensives should be avoided.
- The use of sedative and hypnotic drugs can cause falls, so caution should be taken when prescribing these drugs for the elderly.

1.4.5. Inadequate intake of Calcium and Vitamin D:

Bone is the major reservoir for Calcium accounting for 99% of total body Calcium.

The skeletal content of elemental Calcium at birth increases 40-fold by the time skeletal maturity is reached. The recommended daily allowance of Calcium for healthy women varies from country to country and ranges from 400 to 1500 mg daily.

It should be known that accretion of Calcium into bone occurs after matrix production and both in adults and during growth. Thus, the skeletal demands for Calcium are governed by the rate of matrix synthesis rather than the other way round. If the skeletal demands for Calcium are



not met, then hypocalcaemia and defective mineralization of bone will follow.

Low serum Calcium levels will increase Parathyroid hormone secretion and the synthesis of Calcitriol which in turn will increase bone turnover and contributes to the development of Osteoporosis.

The richest source of Calcium is milk and dairy products, other sources include green vegetables, nuts and certain fish. Bioavailability of Calcium from food is 30%.

It is thus clear that without Calcium there would be no skeleton, or at least it would be not mineralized.

Vitamin D is derived from the diet and from the skin by ultra-violet irradiation of 7-dehydrocholesterol. Vitamin D is fat soluble and absorbed primarily from the duodenum and Jejunum into the lymphatic circulation. It is distributed in the fat and muscle.

Before exerting its effects, it undergoes a series of metabolic conversions, the first step involves its conversion in the liver to a 25-hydroxylated derivative, the second step is further hydroxylation mainly in the kidney to 1,25-dihydroxy vitamin D₃ (Calcitriol).

Its principal effects are to increase intestinal absorption of Calcium and phosphate. Several studies have shown that estrogen stimulates the synthesis of Vitamin D.

The recommended daily intake of Vitamin D for children and adults is 200-400 IU, while for over 50 years old people, it is 600-800 IU.

1.4.6. Co-Morbid diseases:

1.4.6.1 Endocrinal causes:

1.4.6.1.1 Hyperparathyroidism:

Is broadly defined as an increase in the circulating level of Parathyroid hormone (PTH) which occurs secondary to increased secretion from the parathyroid cells leading to an increase in the serum Calcium. This occurs in primary hyperparathyroidism.

In secondary hyperparathyroidism, the increase in PTH occurs secondary to a reduction in the plasma concentration of ionized Calcium.

Primary hyperparathyroidism results in impaired bone quality due to an increase in the activation frequency of bone remodeling.

1.4.6.1.2 Thyrotoxicosis:

Osteoporosis occurs in long standing Hyperthyroidism and patients show an increase in the rate of bone remodeling. Hypercalcuria is common but hypercalcaemia occurs rarely.



The mechanism is thought to be due to a direct action of the thyroid hormone to increase bone turnover.

Measurement of thyroid hormones should be done for elderly people with Osteoporosis to correct any hormonal imbalance.

1.4.6.1.3 Cushing's Syndrome:

Glucocorticoids have adverse effects on the skeletal metabolism. Cushing's syndrome is associated with progressive Osteoporosis and fractures.

Usually this syndrome is associated with other features such as Hypertension, centripetal obesity, atrophic changes in the skin and Diabetes Mellitus.

1.4.7. Clinical disorders associated with Osteoporosis:

1.4.7.1 Neoplasia affecting the skeleton:

Osteoporosis is a common complication of several neoplastic disorders. Solid tumours commonly produce focal osteolytic disease, many of them also induce a generalized increase in bone resorption particularly in the case of breast cancer.

Generalized Osteopenia may be a presenting feature of Myelomatosis. Vertebral fractures are present in 50% or more of individuals at presentation. Myelomatosis induces Osteolysis secondary to marked osteoclastic activity by cytokines secreted by the abnormal plasma cells.

1.4.7.2 Liver Disease:

Chronic liver disease especially primary liver cirrhosis is associated with Osteoporosis. It is likely that liver impairment contributes to the Osteoporosis associated with alcohol abuse and haemochromatosis.

Assessment of such patients shows low serum phosphate, low total serum Calcium and high serum activity of alkaline phosphatase.

1.4.7.3 Osteoporosis and Rheumatic diseases:

Several factors have emerged as important determinants of bone mass in patients with Rheumatoid Arthritis. These include age, menopausal status, reduced mobility, disease activity, disease duration and Anti-rheumatic drugs especially Corticosteroids. All the above lead to increased risk of vertebral as well as hip fractures.

Generalized bone loss is also a significant clinical problem in patients with Systemic Lupus Erythematosus (SLE), leading to reduction in both cortical as well as trabecular bone mass.

In Ankylosing Spondylitis, several studies have documented an increased incidence of spinal compression fractures. Because of the paraspinal calcifications and syndesmophytes, many of these fractures are not detected.

1.4.8. Life style:

1.4.8.1 Sedentary life style:

It has long been assumed that adequate physical activity is associated with the build up of a peak bone mass as well as the prevention of osteoporotic fractures.

Athletes in general have greater bone mass and bone mineral density than similar less active persons.

Absolute immobilization causes osteopenia, and if continued it will eventually lead to Osteoporosis.

Postmenopausal women enrolled in a regular exercise program have gained bone.

Simple activities such as walking are useful and can be added to the daily routine of any individual, as it reduces the risk of falls, trauma from falls and fractures. Back strengthening exercises are also useful.

1.4.8.2 Excessive Alcohol intake:

Alcoholics have less bone than controls, and some alcoholics have severe Osteoporosis without any apparent cause. They are also more likely to fall and fracture.

1.4.8.3 Smoking:

Smokers are more prone to Osteoporosis and fractures.

Smoking causes damage of the estrogen receptors and is associated with earlier menopause, however smoking is also a risk factor for Osteoporosis in men as other mechanisms are presumably involved.

1.4.8.4 Excessive caffeine intake:

Osteoporosis has been linked to excessive caffeine ingestion, which has a calciuric effect. Studies have shown that a high caffeine intake was associated with decreased cortical thickness and higher fracture rates in postmenopausal women.

1.4.9. Existing fragility fracture:

The occurrence of fragility fracture is a pivotal point in Osteoporosis, being the hallmark of actual decrease in bone strength, in symptomatology of the condition and in the definite need for active treatment.

1.4.9.1 Fragility fractures:

Fragility fractures are those commonly seen in the osteoporotic patients and are caused by low energy trauma that are not usually associated with fracture in individuals with normal bone strength. These fractures mainly affect sites with high trabecular bone content such as vertebrae, the distal radius, proximal femur including trochanteric



and femoral neck fractures. Fissure fractures of the ribs and pubic rami may cause sudden enigmatic chest or groin pain, which is hard to diagnose and document by x-rays when Osteoporosis is overlooked.

1.4.9.2 Osteoporotic fractures:

Osteoporotic fracture may be defined as any fracture occurring in an osteoporotic bone whether the trauma is of low or high energy, and whether the location is trabecular or cortical. When the trauma energy is high, the bone might as well break in any of the standard ways affecting normal population, but the severity of the fracture is usually higher showing comminution, double level, long segment, or multiple fractures.

Fractures in osteoporotic bone are usually more difficult to treat, especially in internal fixation situations, for metallic screws do not hold well and may become loose early, the bone may take longer time for solid healing. Also, the elderly patient cannot follow the usual rehabilitation programs of partial weight bearing using crutches, which demand relatively strong arms and shoulders, ...etc.

Special techniques may be required when an osteoporotic patient needs other bone operations like total joint replacement, where complications related to poor bone

strength may contribute to higher incidence of failure through the occurrence of periprosthetic fractures and/or early loosening of the implant.

1.4.10. Drugs inducing Osteoporosis:

Bone loss occurs when there is an imbalance between osteoclastic and osteoblastic function. In general, drugs can interfere with bone regulation by one of three mechanisms:

- 1) An increase in osteoclast activation and induction of a high bone turnover state.
- 2) A direct suppression of osteoblastic formation.
- 3) Inhibition of normal mineralization.

Physician awareness, appropriate investigation, careful prescribing, monitoring and proper therapy for this eminently preventable side effects can preserve bone in those patients.

If drug-induced Osteoporosis is diagnosed, clinicians should consider discontinuing the medication identified as the cause. However, in certain cases, discontinuation of the medication may not be feasible. Vitamin D, Calcium and bisphosphonates are the mainstay of therapy for drug-induced Osteoporosis.

Summary of drugs identified to cause Osteoporosis

Drugs	Mechanisms of drug-induced Osteoporosis
<i>Corticosteroids:</i>	↓ Intestinal absorption of Calcium
Oral	↓ Osteoblastic function and maturation
Parenteral	↑ Hypercalcuria
Inhaled } Topical }	Promotion of apoptosis of osteoblasts and osteocytes
<i>Hormonal agents:</i>	↓ Gonadotropins, estradiol and testosterone production and secretion
Medroxyprogesterone acetate	↑ Bone resorption
Leutinizing Hormone Releasing Hormone (LHRH) agonists	
<i>Thyroid hormone replacement therapy: L-thyroxine</i>	↑ Activation of bone remodeling units ↓ 1,25 (OH) ₂ D and parathyroid hormone synthesis
<i>Anticoagulants:</i>	↑ Osteoclastic activity
Heparins	↓ Osteoblastic activity
<i>Anticonvulsants:</i>	↑ Inactive vitamin D metabolites
Phenytoin, carbamazepine (Tegretol)	↓ Intestinal Calcium transport, secondary Hyperparathyroidism.
Phenobarbital, primidone	
<i>Psychotropic drugs:</i>	Inhibition of osteoblastic activity
a. Neuroleptics	↑ Calcium mobilization from the bone
b. Lithium	Hyperparathyroidism, Hypercalcaemia
<i>Exchange resins:</i>	↓ Absorption of vitamin D
Cholestyramine	

Table 2

1.4.10.1 Corticosteroids:

Corticosteroids cause a dose and duration-dependent Osteoporosis, with especially hazardous toxic effect in the elderly.

Prolonged administration of corticosteroids is a common cause of Osteoporosis in adults and stunted skeletal growth in children.

The mechanism of Corticosteroid-Induced Osteoporosis (CIO) is multifactorial. This includes the effects on Calcium homeostasis, bone formation and resorption, and sex hormones:

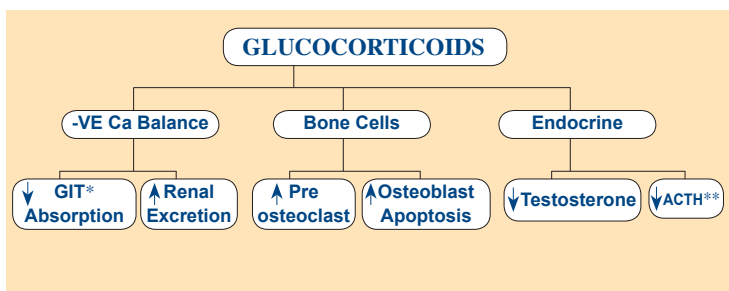


Figure 05

* Gastro Intestinal Tract.

** Adreno Corticotrophic Hormone.

Effect on Calcium homeostasis:

They inhibit intestinal absorption of Calcium and increase urinary Calcium excretion. By creating a net negative



Calcium balance in the body, corticosteroids induce a secondary hyperparathyroid state, thereby stimulating expression of PTH receptors in osteoblasts and enhancing osteoblast responsiveness to PTH. Corticosteroids, also appear to decrease osteocalcin levels, therefore decreasing bone formation.

1. Inhibition of bone formation and enhanced bone resorption: Corticosteroids promote the proliferation and differentiation of osteoclast precursors stimulating increased osteoclast formation. Corticosteroids also promote osteoblast apoptosis and directly inhibit the synthetic function of osteoblasts, causing decreased osteoblast maturation and collagen formation.

2. Effect on sex hormones:

Corticosteroids appear to have both a direct effect on end-organ production of testosterone and suppression of Adreno Corticotrophic Hormone (ACTH) causing decreased gonadotropin production.

There is no evidence to support a dose threshold below which corticosteroids have no effect on bone metabolism. The risk of CIO may be related to cumulative dose of corticosteroids; even intermittent courses can therefore increase the risk. The rate of steroid-induced bone loss is greatest in the first 3-6 months of therapy, so preventive

measures are imperative for maximal risk reduction. After discontinuing corticosteroids, a rebound of osteoblastic function and new bone formation usually occurs, though bone mass may not reach pre-treatment levels.

Published information states that patients are at risk for developing Osteoporosis if they consume prednisone >7.5 mg daily (or equivalent) for more than three months. However, lower doses may also have a significant impact.

Long-term use of high-dose inhaled or topical corticosteroids also contribute to CIO.

1.4.10.2. Other drugs:

- **Thyroid hormone replacement therapy:**

Hyperthyroidism causes increased bone remodeling by accelerating bone turnover which alters bone Calcium mobilization, leading to a net negative Calcium balance available for bone formation.

In general, it is now believed that replacement and mildly suppressive doses of L-thyroxine are not associated with mineral bone loss. However, in a high risk group, such as postmenopausal women who need to be treated with thyroid replacement therapy, the risk of Osteoporosis may be modified or prevented with estrogen therapy, adequate exercise and Calcium supplementation.

- **Antacids:** They inhibit phosphate absorption and disrupt bone mineralization. Both Aluminum and Lithium interfere with intracellular signaling, Aluminum also impairs osteoblast function and causes osteomalacia
- **Diuretics** producing calculia.
- **Antibiotics** long term or frequently including tetracycline.
- **Chemotherapeutic agents**, e.g.: methotrexate, cyclosporine A (which is also used in transplantation) have been shown to increase bone turnover in rodent studies.
- **Benzodiazepines** including Valium and Xanax.
- **Agonists of gonadotropin-releasing hormone:** long term use of these hormones reduce circulating estrogen levels and thereby cause excessive bone loss.

High-risk groups:

1. Postmenopausal women.
2. Persons above 65 years old.
3. Persons with an existing fragility fracture.
4. Patients receiving Osteoporosis-inducing drugs, e.g.: corticosteroids.

1.5. Diagnosis of Osteoporosis:

Osteoporosis is a silent disease, symptoms do not occur except after occurrence of fractures.

Risk factors that increase the index for suspicion are well defined, physicians should detect and identify people at risk for Osteoporosis and subject them for further investigations, in order to discover such a serious silent disease that could proceed through an insidious course until a major health problem endangers the life of the patient.

Before the occurrence of bone deformities, no clinical signs could be detected.

1.5.1. X-RAY in the diagnosis of Bone Mineral Density (BMD):

X-ray was the available method for diagnosis until late in the 20th century.

X-ray could detect low bone density after the bone loss exceeds about 40% of its mineral content. Also it is a very subjective method of diagnosis that differs from one clinician to another, and not standardized.

In the seventies of the last century, radiological techniques have been developed to detect the Bone Mineral Density of different sites in the body.

1.5.2. Ultrasound in the diagnosis of BMD:

Ultrasound does not measure BMD, but it gives an idea about the BMD. When a part of peripheral bone is exposed to ultrasound beam, this beam will be attenuated by a degree comparable to the density of this particular bone: broad beam attenuation.

This broad beam attenuation is a helpful method for suspecting Osteoporosis, the technique is easier, and cheaper, while its accuracy and reproducibility are suspected.

When Osteoporosis is suspected on the base of ultrasound evaluation, patient must be subjected to DXA examination, to avoid over diagnosis or under diagnosis.

Ultrasound machines usually measure calcaneus or wrist. It is generally a useful tool for screening.



Figure 06

1.5.3. DXA in the diagnosis of BMD:

The Dual photon X-ray Absorbtiometry (DXA) is the approved standard for diagnosis. It estimates the Bone Mineral Content (BMC) of the examined part and consequently, the Bone Mineral Density (BMD), i.e.: BMC in one square centimeter. It is not possible till now to have the real density that should be estimated in cubic centimeter.

The DXA machine is having an X-ray tube that is situated below the patient lying in supine position or sometimes in lateral position. Two photons are generated consequently from the X-ray tube. They pass through the examined part, to be detected by a moving sensor above the lying patient.

The difference in photon densities represents the bone density of the examined part.

The generated X-ray beam could be very sharp and cover a small well-defined specific spot on the examined part, i.e. pencil beam, or it could be fan beam that cover a wider area of the examined part.

The pencil beam technology is a very specific measurement, that is more useful for follow up as we could detect specifically the area that has been measured before, to estimate the changes in the BMD in this particular spot, although it is more time consuming, the scan time may reach 12 minutes for each part of the body. While the Fan beam

covers a wider area and the scan time is faster (three times faster than the pencil beam), but the follow up accuracy is not the same as in pencil beam. Computer technology has developed the time needed for the pencil beam to scan an area of the body.

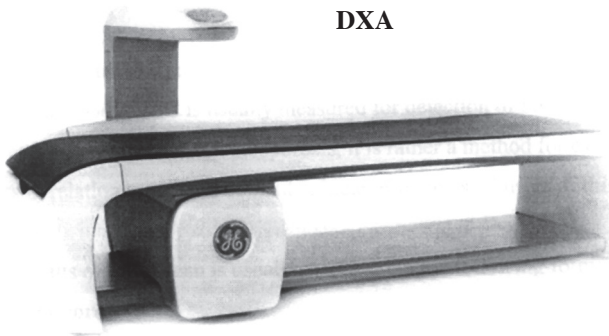


Figure 07

The body sites usually measured are:

A. Femur:

Intertrochanteric area, ward's triangle, femoral neck and total femur.

B. Vertebrae:

Lumbar vertebrae are measured usually in the

Posteroanterior (PA) position. Each vertebra should be measured independently and then results are analyzed to get a conclusion representing their bone density. In elderly people, with calcified great vessels, the lumbar vertebrae may be obscured, hence the necessity of the lateral position to avoid the density of the great vessels that may impede the passage of the X-ray beam.

C. Forearm:

Distal third of radius.

An important point regarding the forearm measurement is that the forearm may be deceiving as it is nonweight bearing, hence it is usually showing low BMD, so we may over diagnose Osteoporosis if we rely on it. The other sites are weight bearing and any fracture in any of the vertebrae or the femur is more risky. Some authorities like to disregard the forearm measurements.

D. The total body:

The whole body BMC is usually measured for detection of body composition, it is not a method for diagnosis of Osteoporosis, it is rather a method for estimating lean body mass in relation to mineral content, athletes are the usual users of this technique.



The results of DXA scan is usually displayed in the following forms:

A. Z-score:

Displays the relation between the measured BMD and the standard BMD for the same age in a similar ethnic group with the same body weight.

B. T-score:

Displays the result of the measured BMD in comparison to the standard BMD of young adult (25 years age) in the same ethnic group and same body weight.

The T-score is the reference score for diagnosis and management, while Z-score is looked for to detect cases of secondary Osteoporosis when the reading is low, even below the comparable age group, so one has to be sure that there is no other cause for Osteoporosis.

C. Absolute figure of BMD:

Usually displayed in separate page, as an ancillary report, showing the actual BMC for each measured part in mg/square centimeter of bone.

1.5.4. Bone markers in the diagnosis of Osteoporosis:

Bones like any organ in the body are biologically active and living, so we can assess the bone state by measuring the metabolites that measure bone building condition or

bone resorption state, and determine which is taking the upper hand.

Markers of bone formation:

- Serum Bone Specific Alkaline Phosphatase: (BSAP).
- Serum Osteocalcin.
- Procollagen type I N-terminal Propeptide: (P1NP).

Markers of bone resorption:

- Urine and Serum N-crosslink Telopeptides: (NTX).
- Urine and Serum C-crosslink Telopeptides: (CTX).

The most commonly used ones are: BSAP and Osteocalcin as bone forming markers and NTX and CTX as markers of bone resorption.

Measurements of bone markers are more costly than measuring BMD, but BMD changes need one year to be detected. Bone markers, as other body fluid solutes, could show earlier changes in bone formation and resorption, to assure the patient and the doctor about the improvement of the patient's condition.

Accurate diagnosis of bone density is needed for the start and follow up of the treatment.

Combining the bone markers measurement and BMD measurement may be more helpful for diagnosis.

- 
- Osteoporosis is not a clinically detectable disease in its early phase unless a fracture occurs.
 - X-ray could detect low bone density when the bone loss exceeds about 40% of its mineral content.
 - Ultrasound gives a rough idea about the BMD, it is generally a useful tool for screening.
 - DXA is the approved standard for diagnosis.
 - We can assess the bone status by measuring the metabolites of bone forming or bone resorption (bone markers), that determine which is taking the upper hand.
 - BMD changes need one year to be detected. Bone markers could show early changes in bone formation and resorption.

II. Egyptian Guidelines for the Management of Osteoporosis

II. Egyptian Guidelines for the Management of Osteoporosis

2.1. Objectives.

2.2. Prevention of Osteoporosis.

2.3. Management modalities.

2.3.1. Non-pharmacological approach.

2.3.2. Pharmacological approach.

2.3.3. Management of osteoporotic fractures:

2.3.3.1. Fall prevention.

2.3.3.2. Pain management.

2.3.3.3. Orthopaedic management.

2.3.4. Monitoring therapy.

2.1. Objectives:

These Guidelines are designed for use by General Practitioners and specialists in their daily practice. It will optimize the benefit gained from the use of the existing therapeutic modalities and will help them in selecting the most appropriate one for their patients based on the best available evidence.

2.2. Prevention of Osteoporosis:

2.2.1. Primary prevention:

Primary prevention aims ideally at improving the strength and quality of bone so it becomes more resistant to fragility fractures. It also addresses decreasing the likelihood of falling accidents by eliminating known factors related to patients- associated medical and residential conditions that may contribute to frequent falling in elderly.

Initiation of active primary prevention is justified in:

- Women at 75 years of age, without the need for prior DXA scan.
- Women between 65 and 74 years, if the presence of Osteoporosis is confirmed by DXA.
- Postmenopausal women < 65 years of age with DXA T-Score between - 1 and - 2.5 SD plus one or more additional age-independent risk factor. (see risk factors).

2.2.2. Secondary prevention:

Secondary prevention is indicated in postmenopausal women who have sustained a clinically apparent osteoporotic fracture.

A postmenopausal woman who has sustained a clinical fragility fracture is a candidate for initiation of active

treatment, regardless of her DXA status, such treatment aims at prevention of further fractures development, which are more likely to develop after the occurrence of the first fracture.

Life style modification:

2.3.1.1. Calcium and Vitamin D (supportive treatment).

2.3.1.2. Exercise.

2.3.1.3. Proteins moderation.

2.3.1.4. Alcohol cessation.

2.3.1.5. Smoking cessation.

2.3.1.6. Caffeine moderation.

2.3.1.7. Carbonated beverages moderation.

2.3. Management modalities:

2.3.1. Non pharmacological approach:

The idea is to prevent the occurrence of Osteoporosis and its consequent fractures. This is achieved mainly through life style modification.

2.3.1.1. Calcium and Vitamin D (supportive treatment):

Calcium and Vitamin D supplementation must be used in combination with all antiosteoporotic drugs as a routine. Patients with advanced renal disease should be given the active form of Vitamin D.

Calcium:

Calcium is essential to ensure proper bone modeling as well as bone remodeling. Calcium plays a major role in attaining a peak bone mass, it is also capable of slowing the rate of bone loss. The recommended daily Calcium intake for an adult is 1000 mg, but this increases in old people up to 1500 mg. The effect of Calcium in the treatment of Osteoporosis appears to be due to a decreased bone turnover. It seems likely that this is related to the small increments induced in serum Calcium and the resulting decrease in PTH and the activation of bone turnover.

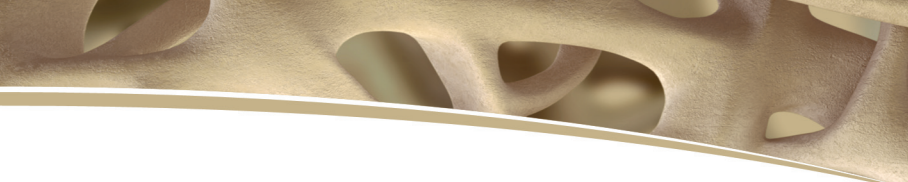
Vitamin D:

This aims at preventing impaired mineralization of bone. Vitamin D deficiency has adverse skeletal effects. It is reasonable therefore that all individuals should have a diet that is adequate in Vitamin D, the recommended daily intake is 400-800 IU (see section 1.4.5.), and where necessary the diet may be supplemented.

The Vitamin D requirements in the elderly may be as high as 1000-1500 IU daily.

2.3.1.2. Exercise:

Regular exercise is important for the general health.



Spending some time exercising outside for 30-50 minutes at least from 3-5 times a week is important. (walking, running, jogging ...etc). Immobility is associated with an increased risk of Osteoporosis and should be avoided in elderly people.

Regular physical activity is recommended for all age groups. Regular exercise is also known to stimulate bone gain and decrease bone loss.

Moderate physical activity in people with Osteoporosis can both improve their fitness and overall quality of life.

Aerobics and non-weight bearing activities such as swimming improve well-being, increase confidence and coordination that may decrease the risk of falls.

With respect to skeletal health, weight-bearing activities such as walking, cycling are beneficial.

2.3.1.3. Proteins moderation:

Normal protein intake is important in the elderly since protein deficiency lowers Insulin Growth Factor –1 (IGF-1).

Adequate protein intake (1 gm / kg body weight) is also important in patients with hip fracture. Well balanced diet reduces morbidity and mortality.

The following points should be considered:

- Healthy diet with adequate minerals, vitamins and proteins.
- Modification of life style.
- Exposure to sunlight.
- Correction of medical conditions known to increase the likelihood of falls, e.g.: vision, increased frequency of micturition, dizziness, ... etc.

2.3.1.4. Alcohol cessation:

Abuse of alcohol should be avoided. Alcoholics have less bone, less absorption and impaired homeostasis of Calcium. Defective synthesis of Vitamin D by the liver and poor nutritional status contribute to bone loss. Needless to say that alcoholics have greater risk to fall.

2.3.1.5. Smoking cessation:

Smoking leads to destruction of estrogen receptors in young women.

Cigarette smoking deprives the body from estradiol and converts some of the normally produced estradiol into an altered form which has no estradiol activity. It also blocks

estrogen receptors and reduces the number of viable follicles in the ovaries.

2.3.1.6. Caffeine moderation:

Regular intake of 3-4 cups of coffee daily induces a negative Calcium balance and increases the risk of Osteoporosis secondary to decreased cortical thickness.

2.3.1.7. Carbonated beverages moderation:

These beverages are hazardous due to its high phosphate content that leads to Hypercalcuria.

Kindly note that:

- Calcium plays a major role in attaining a peak bone mass.
- The recommended daily Calcium intake for an adult is 1000 mg, but this increases in old people up to 1500 mg.
- Individuals should have a diet that is adequate in Vitamin D.
- The Vitamin D requirements in the elderly may be as high as 1000-1500 IU daily.
- Moderate physical activity in people with Osteoporosis can both improve their fitness and overall quality of life.

- Well balanced diet reduces morbidity and mortality.
- Smoking leads to destruction of estrogen receptors in young women.
- Regular intake of 3-4 cups of coffee daily induces a negative Calcium balance.
- The carbonated beverages are hazardous due to its high phosphate content.

Algorithm for the management of postmenopausal Osteoporosis

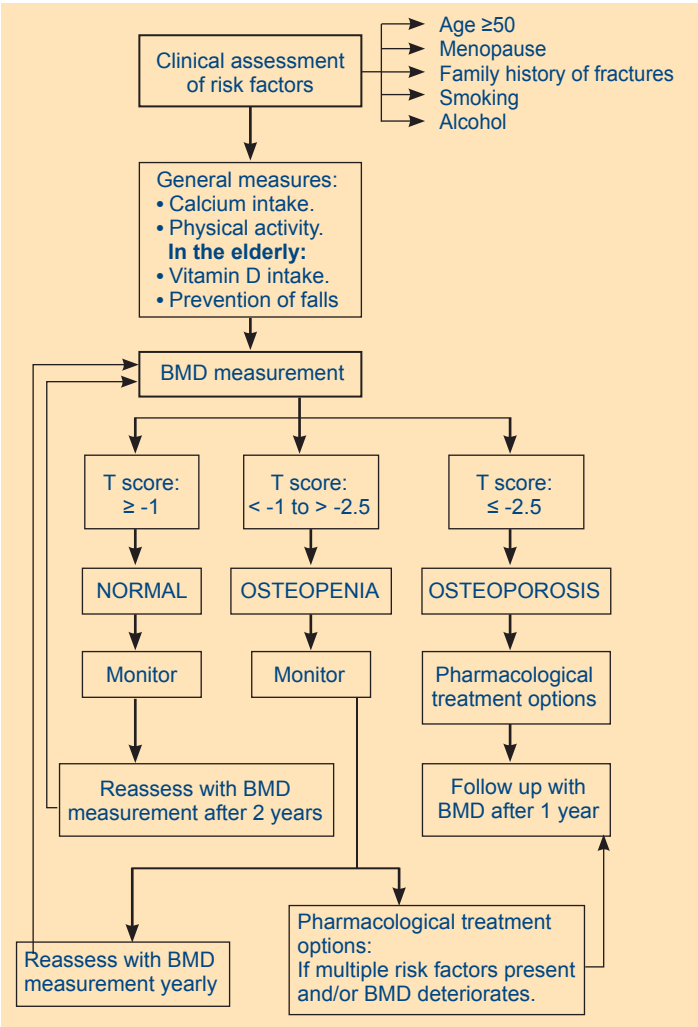


Table 03

2.3.2. Pharmacological approach:

Osteoporotic treatment:

- 2.3.2.1. Hormonal replacement therapy (HRT).*
- 2.3.2.2. Anti resorptives drugs.*
- 2.3.2.3. Bone forming agents.*
- 2.3.2.4. Dual acting bone agents.*

2.3.2.1. Hormonal replacement therapy (HRT):

- Estrogen is a good antiosteoporotic agent, but HRT should be used only for symptomatic relief for the shortest period of time.
- HRT should not be used for cardiac protection (American College of Cardiology ACC).
- HRT should not be given to women with higher risk for **Deep Venous Thrombosis (DVT)**, **Pulmonary Embolism (PE)**, cancer breast, stroke.
- Treatment of Osteoporosis is a long-term treatment and HRT is a short-term treatment hence it is difficult to fit both together.

2.3.2.1.1. Tibolone:

It is a synthetic steroid that exerts favorable tissue selective estrogenic activity on the bone and anti estrogenic activity on the breast, which may help in the management in

menopausal symptoms. However, the molecule is entitled for all the estrogen hazards mentioned in the Women Health Initiative and the One Million Women study as well. There is no available antifracture data for this molecule.

2.3.2.2. *Anti resorptives drugs:*

2.3.2.2.1. *Calcitonin,*

2.3.2.2.2. *Bisphosphonates,*

2.3.2.2.3. *SERMS.*

2.3.2.3. *Bone forming agents:*

Teriparatide.

2.3.2.4 *Dual acting bone agents:*

Strontium Ranelate.

2.3.2.2.1. *Calcitonin:*

Salmon calcitonin is a potent inhibitor of osteoclast activity in vitro. In clinical settings, calcitonin has modest effects on BMD, with values in the hip and spine increasing by 1% to 3% after 3 to 5 years of treatment. Originally given by subcutaneous injection, calcitonin is also administered as a nasal spray.

In women with established Osteoporosis, therapy with nasal calcitonin (200 IU daily for 3 years) has been shown to reduce the risk of new vertebral fractures by 36%, while no effect is observed on nonvertebral fracture risk. Small

studies suggest that calcitonin may fasten the improvement of bone pain following acute vertebral fractures.

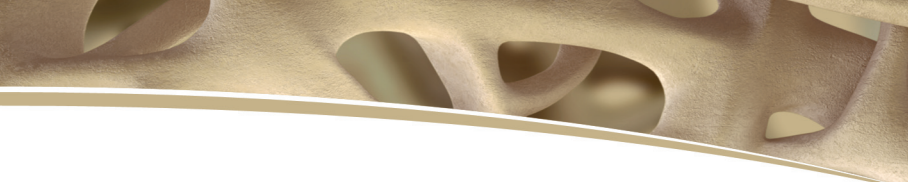
Because calcitonin is a less potent agent than other pharmacologic therapies for Osteoporosis, it is reserved as an alternative for women who cannot or choose not to take one of the other agents. It is recommended for use in women who are at least 5 years beyond menopause because efficacy has not been observed in the early postmenopausal period.

2.3.2.2.2. Bisphosphonates:

Analogues of naturally occurring pyrophosphates, known since 19th century, regulate Calcium level. They may be classified into Nitrogen containing and chloride containing bisphosphonates.

Chloride containing bisphosphonates mediate their antiosteoclastic action through inhibition of ATP, while the more recent Nitrogen containing bisphosphonates mediate their antiresorptive action through mevalonate pathway. They lower the bone turnover to protect the micro-architecture of the bone and increase BMD, leading to increased bone strength and lowering of fracture risk by 48% in vertebrae and 38% in femur in alendronate studies, and by 39% in vertebrae and 26% in femur in risedonate studies.

Bisphosphonates might cause oesophagitis, which may warrant discontinuation of treatment.



Administration and dose regulations: bisphosphonates should be taken early in the morning on empty stomach (daily dose: alendronate 10 mg and residronate 5 mg, or a weekly dose: alendronate 70 mg and residronate 35 mg) with a large glass of water and the patient should not take any food or lie on his back for half an hour following the ingestion of the drug.

2.3.2.2.3. SERMS: Selective Estrogen Receptors Modulators:

Non-hormonal compounds that bind to estrogen receptors in various tissues, showing estrogen agonistic function on the Cardiovascular system and bone but they exert an estrogen antagonistic function on the breast and endometrium. However, they increase the hot flushes and may be responsible for leg cramps, constipation. They should not be prescribed to women with increased risk of (Deep Venous Thrombosis) DVT.

Raloxifene:

Is currently the only SERM licensed for the treatment and prevention of Osteoporosis in post menopausal women, it is available in 60 mg daily tablets.

The MORE study indicates that it reduces the relative risk of the vertebral fracture by 30%, while it did not show

any protection against non-vertebral fractures. It does not increase the incidence of breast cancer like estrogen. Furthermore, it exerts some favorable effects in protection against breast cancer. It may improve the sexual function in elderly women. There are also some opinions that it lowers fibrinogen and both total and LDL cholesterol without increasing HDL cholesterol.

2.3.2.3. Bone forming agents:

Teriparatide:

Is a recombinant human parathyroid hormone acting as an anabolic agent. It stimulates new bone formation. It is also claimed to increase resistance to fragility fracture. The recommended dose is 20 micrograms injected subcutaneously once daily. Patient taking Teriparatide must receive special training on the injection technique. The maximum total duration of treatment is restricted by the license to 18 months in Europe and 24 months in USA.

Particular contraindications include preexisting hypocalcaemia, severe renal impairment, metabolic bone diseases other than Osteoporosis especially hyperparathyroidism and Paget's disease of bone, unexplained increase of the level of alkaline phosphatase and previous radiation therapy to the skeleton.

Special precautions should be undertaken while measuring the serum Calcium and alkaline phosphatase level in patients under treatment with Teriparatide.

The PTH trial indicates that it reduces the relative risk of vertebral fractures by 65% and of the femur by 50%.

The treatment with Teriparatide should be considered in two specific situations:

1) Severe Osteoporosis in patients aged 65 years and older where the prevention of further deterioration of BMD only is thought to be insufficient and stimulation of new bone formation is desirable such as:

1. T- score ≤ -4 SD,

2. T-score ≤ -3 SD + multiple fractures (more than 2) +1 or more of the following additional risk factors:

- Low body mass index < 19 .
- Family history of maternal hip fracture before the age of 75 years.
- Untreated premature menopause.
- Complications associated with prolonged immobility.

2) Unsatisfactory response to first line treatments (Bisphosphonates or Strontium Ranelate).

2.3.2.4. Dual acting bone agents:

Strontium Ranelate:

A new synthesized agent, consisting of 2 atoms of stable Strontium and an organic moiety “ranelic acid”.

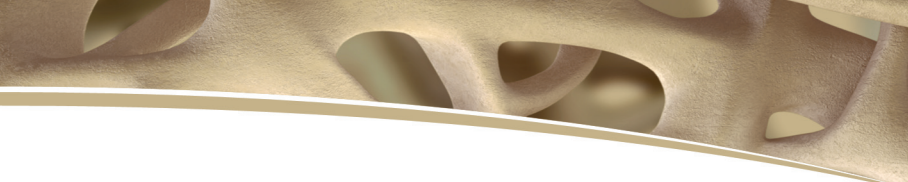
It is a Dual Acting Bone Agent, simultaneously increasing the creation of new bone and decreases bone resorption, thus rebalancing bone turnover in favor of formation. Recent studies have proved that Strontium Ranelate improves Micro architecture and improves bone strength while preserving the proper mineralization of bone.

In the recent SOTI trial, Strontium Ranelate (2 gm/day) orally reduced the relative risk of vertebral fractures by 49% after one year and 41% after 3 years.

The TROPOS trial showed that Strontium Ranelate reduces the relative risk of the hip fractures by 41% over a 3 years period and is well tolerated. It also confirmed that Strontium Ranelate is effective in reducing vertebral fractures.

The drug to be taken: 1 sachet daily, 2 hours apart from dinner (calcium-containing food, Calcium-containing oral medications) to guarantee best GIT absorption.

The most common adverse effects are nausea and diarrhea, generally mild, transient and do not need treatment discontinuation.



Prevention and treatment strategies for Corticosteroid Induced Osteoporosis (CIO):

A risk versus-benefit assessment should always be performed when long-term therapy with corticosteroids is required. There are several options to minimize the effects of corticosteroids on the bone.

These include:

- Discontinue Corticosteroid therapy if possible.
- Minimize exposure to Corticosteroid therapy by using the lowest possible daily dose for the shortest possible time.
- Use alternate-day therapy. However, this method may result in bone losses similar to daily doses.
- Improve Calcium absorption by Calcium and vitamin D therapy.
- Consider using inhaled Corticosteroids whenever possible. It is recommended that supplementation with Calcium Carbonate sufficient to ensure a daily consumption of 1500 mg (or equivalent) daily and vitamin D of 800 IU daily may preserve bone mass in patients receiving long-term treatment of Corticosteroids.

- Inhibit CIO with pharmacotherapy: Bisphosphonates, in addition to vitamin D and Calcium are effective in both prevention and treatment of CIO. Second-line therapy include Hormone Replacement Therapy in women and testosterone in men, calcitonin and thiazide diuretics. Patients who have a urine Calcium excretion > 300 mg/24h may benefit from the addition of a thiazide diuretic (e.g.: hydrochlorthiazide 25mg/day).

2.3.3. Management of osteoporotic fractures:

2.3.3.1. Fall prevention:

Risk factors predisposing the elderly to fall should be detected and treated early:

- Home alteration: improvement of night illumination, removing floor steps and obstacles, addition of side rails and bathroom appliances.
- Modifying the environment to reduce the risk of slipping and tripping by eliminating slippery surfaces, loose rugs, narrow pathways, and dangerous furniture.
- Wearing hip and shoulder protectors.
- Wearing appropriate footwear.
- Taking care when walking up or down stairs.
- Correcting poor vision.

- Modifying medications (e.g.: sedatives, antidepressants or certain antihypertensives that might predispose patients to fall).
- Specific exercise programs that emphasise weight bearing.
- Muscle strengthening and balance retraining.

Most people report trips, slips and loss of balance as the cause of falls, whereas only a small proportion report dizziness or feeling faint.

How to prevent falls:

- Improvement of night illumination, removing floor steps and obstacles.
- Eliminating slippery surfaces, loose rugs.
- Wearing hip and shoulder protectors.
- Appropriate footwear.
- Correcting poor vision.
- Modifying medications.

2.3.3.2. Pain management in osteoporotic fractures:

Conservative treatment is adopted in fissures and non displaced fractures known commonly:

- Vertebral fractures.
- Rib fractures.

Some hip fractures, distal radius fractures, proximal humerus fractures.

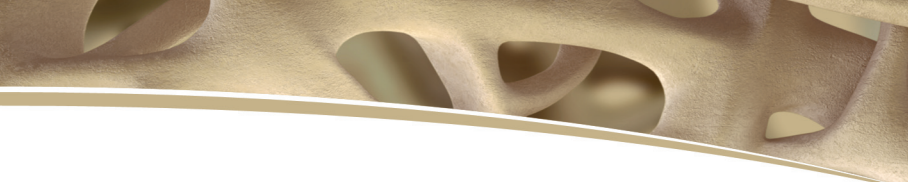
The excessive use of **NSAIDs** (Non Steroidal AntiInflammatory Drugs) for pain control, in this group, may lead to gastric, duodenal and intestinal pathology with its consequent complications. Acetaminophen (Paracetamol), Dextropropoxyphene Hydrochloride and Tramadol Hydrochloride are more suitable for this group as pain control medication provided they are given in adequate doses. Many Physicians suggest use of the Calcitonin for pain control in this situation, assuming it is more specific for bone pain, the cost versus benefit is left to the Physician's judgment to be addressed on individual basis.

Rest is mandatory for fracture pain control, and local rest could be achieved by spinal braces, extension belts, and chest belts. In certain situations a patient may require local infiltration anaesthesia, transdermic phentanyl patches for painful spots or intercostals nerve block for rib fracture pain. Local heat application can also ease the pain, as well as electric methods of pain relief like Transient Electric Nerve Stimulation (**TENS**) and Micro-Current skin patches.

2.3.3.3. Orthopaedic management of osteoporotic fractures:

1. Treatment of back deformity:

- a. Physiotherapy for muscle strength.
- b. Splints and braces.



c. Walking aids.

2. Treatment of vertebral fractures:

- a. Bed rest.
- b. Splints.
- c. Plaster jackets.
- d. Surgical treatment by:
 - Instrumentation and internal fixation.
 - Vertebroplasty (cement injection).

3. Vertebral fractures complicated by neurological problems are treated by decompression.

4. Treatment of hip fractures:

- a. Conservative treatment by traction, splint, ... etc.
- b. Internal fixation using special screws to hold weak cancellous and osteoporotic bone.
- c. Joint replacement.
- d. Partial or total arthroplasty.

5. Treatment of distal radius, proximal humerus, calcaneus and ankle fractures:

- a. Closed reduction and splint.
 - b. Internal fixation by special tools.
- Internal fixation of osteoporotic fractures needs specific surgical techniques and specific materials.

6. Diaphyseal fractures:

- a. Conservative approach.

- b. Open reduction and internal fixation using special tools for bone fixation.
- c. Bone augmentation (cement injection or bone substitutes).

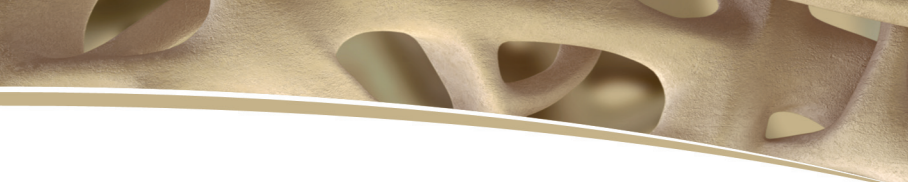
2.3.4. Monitoring of therapy:

Antiosteoporotic treatment is a long term treatment, so the patient may become anxious, and the physician may need some tests or laboratory investigations to ensure the patient improvement in order to motivate him to comply with his treatment. It is advisable that BMD not to be done before one year, after initiation of therapy to avoid patient frustration.

However, bone markers can be done at intervals of 3-6 months to give an idea about the bone status concerning bone formation and resorption. It is imperical to compare the results of the BMD done yearly to judge the response of the patient to the given antiosteoporotic therapy.

Osteoporosis is a disease of rapidly increasing prevalence and high morbidity. Increased awareness of this condition by both the medical community and the public has led to a gratifying reduction in the rate of osteoporotic fractures and improvement in the quality of life of the patients.

During the last decade, several new therapeutic options have emerged, characterized by the unequivocal demonstration of



their anti-fracture efficacy and an improved safety profile, leading to a positive risk/benefit balance. Whereas most of them have proven to significantly reduce the occurrence of vertebral fractures, some discrepancies remain regarding the level of evidence related to their non-vertebral antifracture effect.

The clinical science of Osteoporosis treatment is clearly on the march. We have sensitive methods for early detection of bone loss in high-risk persons, as well as effective treatments such as bisphosphonates, PTH as well as Strontium Ranelate. In the future we can look forward to more potent selective steroid analogues, RANKL antagonists, and anticytokines in addition to growth factors to add to our armamentarium of antiresorptive and anabolic therapies.

*The editorial board aims, by providing this
Guidelines based on
unbiased evidence, to help establish a frame
work for the diagnosis and management
of
Osteoporosis in Egypt.*

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Appendix

Appendix I

Diet and Osteoporosis

A treatment plan for Osteoporosis should ensure sufficient amounts of Calcium and Vitamin D in the diet. Calcium is the major component of bone, and is therefore crucial to maintain bone density. Vitamin D allows Calcium absorption, and its deposition in bone.

Foods rich in Calcium include milk, yogurt, cheese, sardines, salmon, and some green and leafy vegetables such as spinach. The main food sources of vitamin D are fish (e.g.: salmon, tuna), fortified milk, egg yolks, liver and especially fish oils.

Calcium Rich Diet

Food	Quantity	Calcium (mg)
Skimmed or low fat milk	1 glass (245 ml)	295 mg
Chocolate with low fat milk	1 glass (250 ml)	273 mg
Cheddar cheese	28 gm	204 mg
Cottage cheese	½ glass (113 gm)	78 mg
Mozzarella cheese	28 gm	207 mg
Ice cream	½ cup (66 gm)	84 mg
Spinach	½ cup (90 gm)	122 mg
Canned sardines with oil	4 small pieces (48 gm)	183 mg
Almonds	14 gm (7 almonds)	35 mg
Orange	1 fruit	58 mg
Tofu steamed	100 gm	510 mg

Table 01

Foods Interfering with Calcium Uptake:

Some food interfere with Calcium availability. Excessive protein, sodium and caffeine can increase the urinary excretion of Calcium, while excessive fiber can interfere with its absorption.

Food high in oxalic acid (oxalates), such as spinach, rhubarb, beet greens and almonds bind the Calcium and make it unavailable. Legumes such as beans and peas are high in phytates, another substance that interferes with Calcium uptake. These can be soaked for several hours in water, rinsed and cooked in new water to neutralize the effect. The National Osteoporosis Foundation recommends eating foods with oxalic acid and phytates one hour before or two hours after the Calcium-rich foods.

Vitamin D Rich Diet

Food	International Units (IU) per serving
Cod liver oil, (1 tablespoonful)	1,360
Salmon, cooked, (100 gm)	360
Mackerel, cooked, (100 gm)	345
Tuna fish, canned in oil, (90 gm)	200
Sardines, canned in oil, drained, (50 gm)	250
Milk: non-fat, reduced fat, whole, vitamin D fortified, (1 cup)	98
Egg, 1 whole (vitamin D is found in egg yolk)	20
Liver, beef, cooked, (100 gm)	15

Table 02

Temporal Requirements of Calcium and Vitamin D

Daily Adequate Intake (AI) for Calcium and Vitamin D

Birth–6 months

210 mg Calcium

200 IU vitamin D

6 months–1 year

270 mg Calcium

200 IU vitamin D

1–3 years

500 mg Calcium

200 IU vitamin D

4–8 years

800 mg Calcium

200 IU vitamin D

9–18 years

1,300 mg Calcium

200 IU vitamin D

19–50 years

1,000 mg Calcium

200 IU vitamin D

51–70 years

1,200 mg Calcium

600 IU vitamin D

71 and older

1,200 mg Calcium

800 IU vitamin D

Pregnant & Lactating

14–18 years

1,300 mg Calcium

200 IU vitamin D

19–50 years

1,000 mg Calcium

200 IU vitamin D

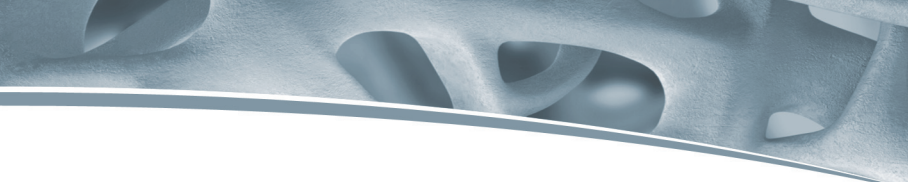
Higher Calcium intake is recommended for certain populations:

- **Postmenopausal women** experience bone loss and decreased Calcium absorption with a decline in estrogen production.
- **Lactose intolerant individuals** do not break down milk sugar in the digestive track, and are therefore at a greater risk of Calcium deficiency due to the avoidance of dairy products.

- **Vegetarians** who only eat plant-based foods probably need more Calcium than that recommended for the average person.

As with Calcium, certain populations are more at risk for vitamin D deficiencies and need more than the recommended dosages:

- **Breast-fed infants** whose only source of food is mother's milk cannot get the proper levels of vitamin D. This lack can be compensated by infant formulas, which are fortified with vitamin D.
- **Adults over 50** are at a higher risk of deficiency due to the decreased ability as we age to produce vitamin D from sunlight, which is the major source for human beings.
- **Homebound people and those living in nursing homes** have less sun exposure and likewise should consider taking supplements.
- **People with fat malabsorption** typically suffer from low levels of vitamin D because it is fat soluble and depends on dietary fat for absorption. Pancreatic enzyme deficiency, Crohn's disease, cystic fibrosis, celiac disease and liver disease are all conditions associated with fat malabsorption.



The need for Calcium and vitamin D supplements:

Food is the preferred source, but if you can't meet your daily requirement from food alone, supplements are often advised.

Calcium supplements derived from bone meal, dolomite, or unrefined oyster shells may contain lead or other toxic metals, so it is best to avoid them.

Calcium Citrate is preferable to Calcium Carbonate as it does not require stomach acid for assimilation. Since the “Calcium load” at any given time is 500 mg, beyond which the body cannot optimally absorb any more, it is recommended that it be taken in increments throughout the day. For optimal absorption, it has been suggested that it is best to take Calcium citrate before bed, and Calcium carbonate at dinner time.

An estimated 30-40% of adults over 50 have a vitamin D deficiency, which accelerates bone loss and increases the risk of fractures. If the patient vitamin D consumption from food is too low, you might consider giving him a Calcium supplement that contains vitamin D, or taking vitamin D alone in supplement form. Vitamin D supplements typically come in strengths from 200 – 400 IU.

Appendix II

Fall Prevention Tips

Occupational health recommendations are often given as a set of tips that help elderly people to alter the home environment in a way that minimizes the likelihood of falls.

The following tips are helpful to follow:

- Ensure that bathtub is not slippery by using rubber mats or non-skid decals.
- Equip home with good lighting. In the middle of the night, it helps to have a well-lit path to the bathroom (i.e.: use night-lights).
- Ensure that dresses, skirts and pyjamas are not too long to avoid tripping over them.
- Secure all loose rugs to avoid slipping.
- Secure all wiring and electrical cords away from common traffic areas and remove clutter.
- Be aware of the potential to trip over pets.
- Falls frequently occur on stairs. Installing stable handrails, ensuring proper lighting (especially at the top and bottom) and wearing appropriate footwear can help prevent falls. Take your time going up and down stairs.
- Cover slippery steps with gritty, waterproof paint.

Modifying activities of daily living, so the performance of everyday activities is achieved with minimized risk for vertebral fractures that could be produced by minimal stress in osteoporotic patients.

- When objects are heavy, it is best if they are positioned at waist height before you attempt to lift them. However, if the object is on the ground, bend your knees and try to keep your spine straight while you bring the object as close to you as possible. Once in this position, use your legs to lift while continuing to keep your spine as straight as you can. (**Figure 1**)



Figure 01

- When getting out of bed, roll over onto one side first and then use your arm to help you sit up. Avoid

getting out of bed by sitting straight up from a position of lying on your back. (Figure 2)

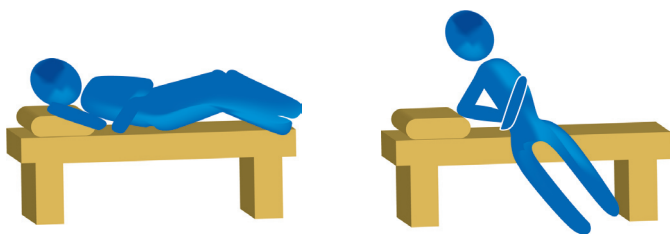


Figure 02

Hip and shoulders protectors:

The role of hip protectors was emphasized in the last ten years, but accumulating evidence cast major doubts about the validity of this assumption, and consequently we do not see hip protectors as a useful tool.

Authors' conclusions:

Accumulating evidence casts some doubt on the effectiveness of the provision of hip protectors in reducing the incidence of hip in older people. Acceptance and adherence by users of the protectors remain poor due to discomfort and practicality.

Appendix III

Common Fragility Fractures, Radiological examples

1- Vertebral Fracture

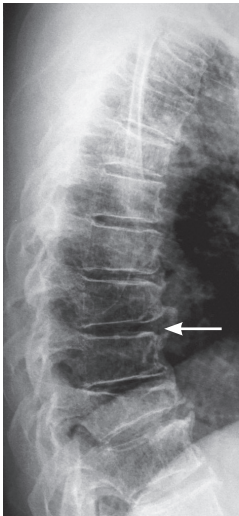


Fig. 1a
Single vertebral fractures

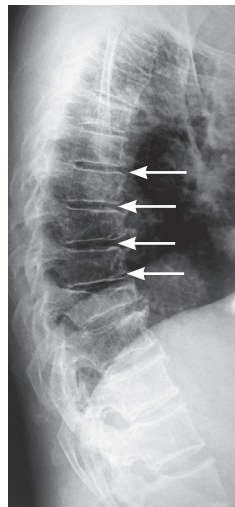


Fig. 1b
Multiple vertebral fractures

2- Hip Fractures

A- Trochanteric Fracture

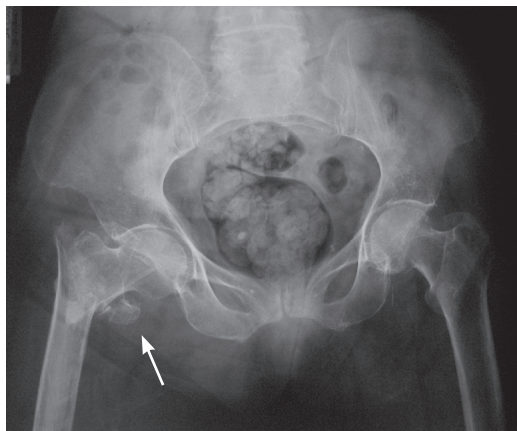


Fig. 2a
Right inter trochanteric fracture

B- Subcapital Fracture

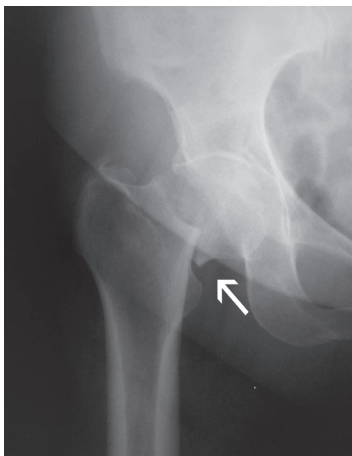


Fig. 2b
Right transcervical fracture

3- Distal Radius Fracture



Fig. 3a, 3b
Lateral and Anteroposterior
views of Colles's fracture.