زانکۆی سەلاحەدین-هەولێر

Salahaddin University – Erbil

Role of Vitamin D in Osteoporosis

A Research Project Submitted to the Council of the College of Education-Shaqlawa, Salahaddin University – Erbil in Partial Fulfillment of the Requirements for the Degree of Bachler in Biology

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بسم الله الرحمن الرحيم

CERTIFICATE

This research project has been written under my supervision and has been submit-ted for the award of the **BSC**. Degree in **Biology** with my approval as a supervisor.

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Date: **14/4/2022**

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Signature: Name: **Sherko muhammed abdul-rahman Head of the department of biology**

Date: **14/4/2022**

I confirm that all the requirements have been fulfilled

DEDICATION

This Research Project is dedicated to: Allah Almighty, my Creator and my Master ,my great teacher and messenger, Mohammed (May Allah bless and grant him), who taught us the purpose of life,My homeland Kurdistan, the warmest womb,The University of Salahaddin-Erbil; my second magnificent home; My great parents, who never stop giving of themselves in countless ways, My beloved brothers and sisters ,To all my family, the symbol of love and giving ,My friends who encourage and support me , all the people in my life who touch my heart.

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Sammary

Vitamin D3 (cholecalciferol) sufficiency is essential for maximizing bone health. Vitamin D enhances absorption of calcium and phosphorus from small intestine that leading to cause increases the plasma levels of calcium and phosphorus. In bone metabolism, vitamin D regulates osteoblast and osteoclast activity, and inhibits PTH hypersecretion, promoting bone formation and preventing/treating osteoporosis. This evidence is supported by most clinical study. The consequences of vitamin D deficiency are secondary hyperparathyroidism and bone loss, leading to osteoporosis and fractures. The major source of vitamin D for both children and adults is exposure of the skin to sunlight. Solar ultraviolet B photons are absorbed by 7-dehydrocholesterol in the skin, leading to its transformation to previtamin D3, which is rapidly converted to vitamin D3. Vitamin D3 is metabolized in the liver to 25-hydroxyvitamin D3 and then in the kidney to its biologically active form, 1,25dihydroxyvitaminD3. Season, skin pigmentation, sunscreen use, clothing and aging can dramatically influence the synthesis of vitamin D in the skin. Very few foods naturally contain vitamin D or are fortified with vitamin D and vitamin D supplementation may decrease bone turnover and increase bone mineral density. Vitamin D deficiency can be prevented by sensible sun exposure and adequate supplementation. Vitamin D status is related to bone mineral density and bone turnover. Monitoring serum 25-hydroxyvitamin D is the only way to determine vitamin D status.

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Introduction

Vitamin D is essential for maintaining calcium homeostasis and optimizing bone health. Vitamin D is a steroid hormone direct action on osteoblasts and osteoclasts and interaction with nonskeletal tissues such as extraosseous tissues, parathyroid hormone (PTH), and the intestine help in maintaining a balance between bone turnover and bone growth (Dawson-Hughes and Bischoff-Ferrari 2007). Vitamin D receptors (VDRs) are distributed on osteoblasts, osteoclasts, and osteocytes and regulate bone remodeling to maintain bone health. he active vitamin D metabolite, binds to a specific vitamin D receptor thereby increasing intestinal calcium absorption and regulating bone turnover (Holick 1999).

Low concentrations of vitamin D lead to alterations in calcium and phosphorus homeostasis, bone loss, osteoporosis, and an increase in fracture risk (Kinyamu, Gallagher et al. 1997, Holick 2004). Adequate vitamin D and calcium intake is considered an essential component of osteoporosis management. The American Society of Bone and Mineral Research suggest that vitamin D along with calcium supplementation is a fundamental treatment for osteoporosis (Rachner, Khosla et al. 2011).

Currently in the United States , the recommended daily vitamin D intake is 400 IU for individuals aged 51–70 year and 600 IU for those aged 70 year and older (Holick 1998). In Europe, 400 IU is recommended for people aged 65 year or older. Although vitamin D is produced in the skin with exposure to sunlight, there is an age-related decline in cutaneous synthesis making older individuals more dependent on dietary intake (MacLaughlin and Holick 1985), and have indicated that vitamin D inadequacy is a common problem

worldwide (Lips, Duong et al. 2001). Differences in the prevalence of vitamin D inadequacy have been related to a variety of factors, including physiological changes with age, race, body mass index (BMI), sun exposure, latitude, and dietary vitamin D intake. Several studies have shown that serum concentrations of at least 20–30 ng/ml are necessary to maximize intestinal calcium absorption (Chapuy, Preziosi et al. 1997, Heaney, Dowell et al. 2003).

the term, osteoporosis, refers to a histological assessment of bone by a pathologist, and it is defined by diminished quantity of bone mineral per unit volume of whole bone. Osteoporosis was first discovered by John Hunter, a British surgeon, in 1800's and he was also the first to introduce the process of remodeling. Jean Lobstein, a French pathologist during 1830's, found that there are normal holes in every bone but bones in people with specific age and diseases, have holes of larger than normal size.

He named this kind of bone as *porous*, and the disease was named as *osteoporosis*. Osteoporosis is a common and silent disease until it is complicated by fractures that become common. It was estimated that 50% women and 20% of men over the age of 50 years will have an osteoporosis-related fracture in their remaining life. These fractures are responsible for lasting disability, impaired quality of life, and increased mortality, with enormous medical and heavy personnel burden on both the patient's and nation's economy. Osteoporosis can be diagnosed and prevented with effective treatments, before fractures occur. Therefore, the prevention, detection, and treatment of osteoporosis should be a mandate of primary healthcare providers. The biggest problem created by bone disease, especially osteoporosis, is fractures, which may be the first visible sign of disease in

patients. Each year an estimated 1.5 million individuals suffer a fracture due to bone disease (Alem, Sherrard et al. 2000).

According to existing literature, vitamin D deficiency is a global problem with various symptoms and major consequences. It does not only affect the bone quality but also increases the risk of fractures, autoimmune diseases, and cancer. The true incidence of osteoporosis, the disease of weakened bones that causes heightened risk of bone fractures. Osteoporosis itself has no symptoms; its main consequence is the increased risk of bone fractures(Shinchuk and Holick 2007, Wacker and Holick 2013).

2 Review of Literature

2.1 Synthesis of vitamin D and metabolism

Vitamin D is a fat-soluble vitamin which would be formed in the skin after Sunlight ultraviolet B radiation (wavelength, 290 to 315 nm) penetrates the skin and converts 7-dehydrocholesterol that to previtamin D3 which is rapidly converted to vitamin D3 (Al-Amin, Thomson et al. 2006). Because any excess previtamin D3 or vitamin D3 is destroyed by sunlight, excessive exposure to sunlight does not cause vitamin D3 intoxication (Mori and Zhou 2016). However a small percentage of total circulating vitamin D also originate from dietary intake and is primarily found in foods such as fatty fish, eggs and milk (Dawson-Hughes and Bischoff-Ferrari 2007). Vitamin D from the skin and diet is metabolized in the liver to 25-hydroxyvitamin D, which is used to determine a patient's vitamin D status (Holick 2004). Hepatic 25-hydroxylase converts cholecalciferol (vitamin D3) to 25(OH)D3, and the vitamin D binding protein carries 25(OH)D to the proximal tubule of kidney. The 1-α-

hydroxylase in the kidney transforms the 25(OH)D3 into 1,25(OH)2D, which serves as the active form of vitamin D with biologic effect. 24-hydroxylase, which converts 25(OH)D to 24,25(OH)2D, decreases the serum 25(OH)D and the active 1,25(OH)2D. The renal production of 1,25-dihydroxyvitamin D is tightly regulated by plasma parathyroid hormone levels and serum calcium and phosphorus levels. (Lips, Duong et al. 2001, Holick 2004). The bioavailability of vitamin D depends on its intestinal absorption capacity, liver health of the individuals, and their fat storage. Adipose tissue easily absorbs vitamin D ingested or produced by chemical affinity. Some authors suggest that the accumulation of vitamin D in adipose tissue is important for its subsequent release during times of reduced production (for example, during winter when the fat storage decreases) (Holick and Chen 2008).

Figure 1. Schematic representation of admission and metabolism of vitamin D. Vitamin D is supplied by cutaneous synthesis or diet intake. The bloodstream takes it into the liver, where its chemical structure is changed by hydroxylation. Then it is sent to the kidneys for another hydroxylation. Finally, the active metabolite 1,25(OH)D circulates through the body in order to be effective. This graphic has been drawn up based on the schematic representation created by Shinchuk 2007 and Heath 2006 (Al-Amin, Thomson et al. 2006, Shinchuk and Holick 2007).

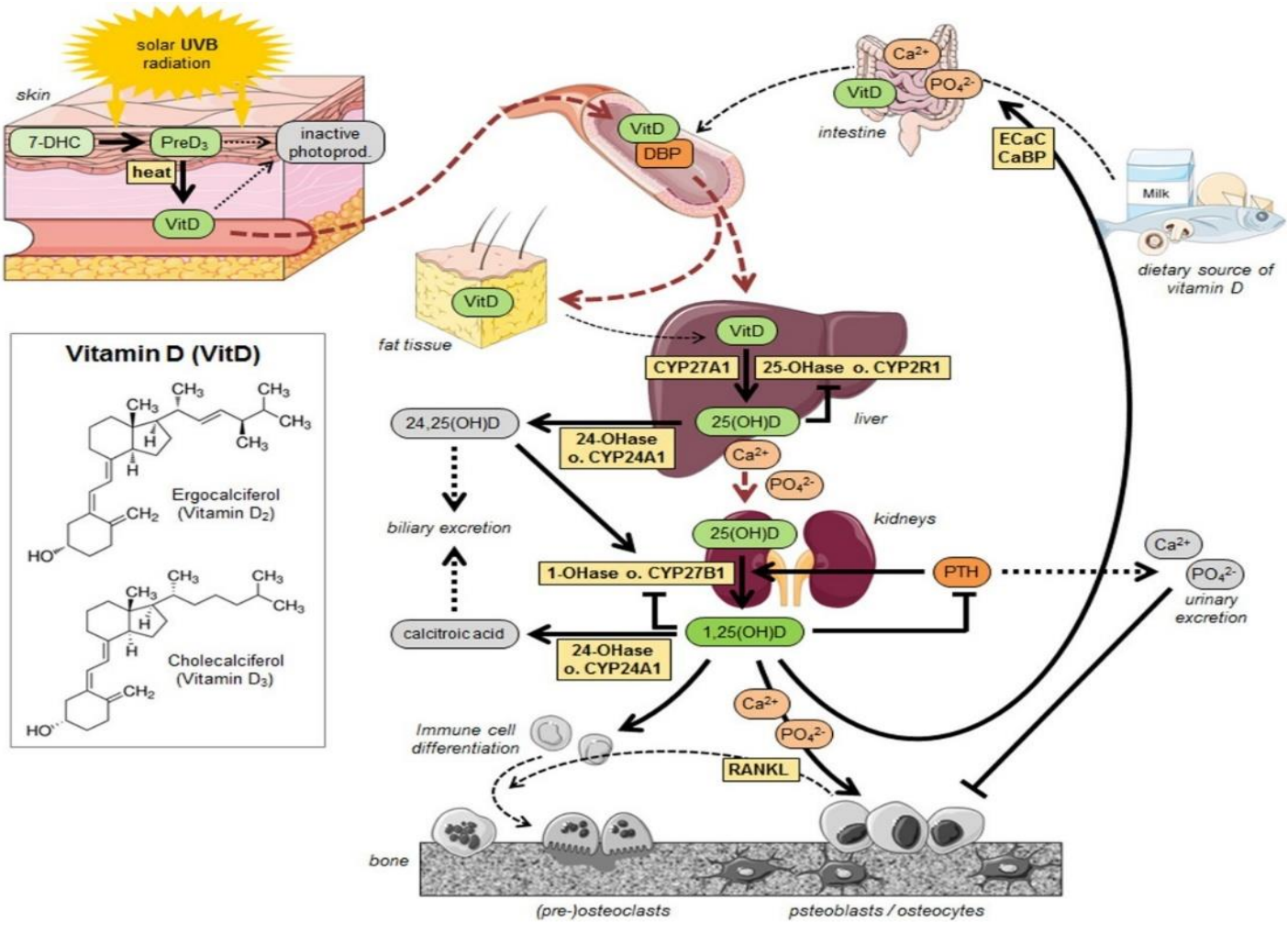

Figure 1:Schematic representation of admission and metabolism of vitamin D.

2.2 Vitamin D and skeletal pathophysiology

The mechanical integrity and structure of skeleton is maintained by the constant remodelling of bone which O. Sahota (Figure 2) responds to the normal physiological and pathological skeletal stresses of daily living. The required intakes of calcium and vitamin D increase with age, which unfortunately, these increased levels are seldom achieved

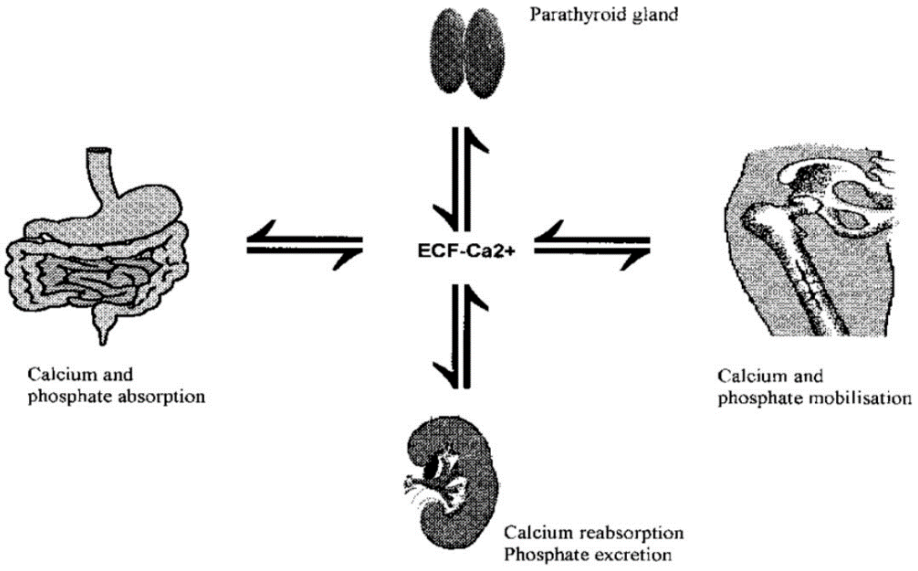

Figure 2:The parathyroid hormone–vitamin D axis

Figure 2. The parathyroid hormone–vitamin D axis.

Vitamin D and calcium are important in the mechanical and structural integrity of the skeleton .

• Subclinical vitamin D deficiency (vitamin D insufficiency) is common in the fit, active elderly population and leads to an amplification of age- related bone turnover, bone loss and thus increased risk of fracture, mediated by secondary hyperpara-thyroidism.

• Daily supplementation with vitamin D can reduce the secondary hyperparathyroidism and increase bone marrow density but only combination calcium and vitamin D therapy has been shown to be effective in reducing non-vertebral fractures, showing that in 2012, about 80% of the European

inhabitants had a vitamin D serum concentration below 30 ng/mL (Zadeh and Kor 2014, Gunathilake and Rupasinghe 2015). The probability of suffering from vitamin D deficiency increases with age (Gunathilake and Rupasinghe 2015). Vitamin D insufficiency and deficiency can occur in various ways. Causal to the deficiency can be, among other things, the reduced intake with diet, inadequate sun exposure or a metabolic disorder (Heath and Elovic 2006, Zhang, He et al. 2015). Risk factors to developing vitamin D insufficiency or deficiency are: (a) reduced or restricted sun exposure (e.g., homebound persons, people covering their skin for cultural/religious reasons); (b) a reduced cutaneous synthesis (e.g., in elderly); (c) suffering from a malabsorption syndrome (e.g., Crohn's disease); and/or (d) obesity (Heath and Elovic 2006, Shinchuk and Holick 2007, Zhang, He et al. 2015). There is no generalized recommendation for the time of sun exposure needed to obtain sufficient vitamin D activation as this process is strongly dependent on skin pigmentation, area of exposure and UV strength. Furthermore, vitamin D is fat soluble and gets easily accumulated in fat–tissue(Shinchuk and Holick 2007, Zhang, He et al. 2015). What must not be neglected is the drug-induced decrease of vitamin D (e.g., glucocorticoids) (Shinchuk and Holick 2007) Symptoms of rickets include changes in bone (e.g., deformities of the leg), a swelling of the wrist with a widened growth gap, a delayed closure of the fontanelles, craniofacial dysmorphy and musculoskeletal pain. Further symptoms like a cardiac arrest, a tetany or seizures might be induced by the resultant hypocalcaemia (Munns, Shaw et al. 2016). Osteomalacia, another metabolic bone disease mainly caused by malfunction of the vitamin D or phosphate metabolism, leads to a reduced bone mineralization in adults (Reuss-Borst 2014) (Rader, Corsten et al. 2015). Unlike rickets, osteomalacia is rare in children (Munns, Shaw et al. 2016). However, there is no specific

blood parameter to prove the presence of osteomalacia. Hence, osteomalacia can be diagnosed on the basis of decreased serum calcium or phosphate levels and an elevated alkaline phosphatase (Reuss-Borst 2014, Fukumoto, Ozono et al. 2015, Rader, Corsten et al. 2015). On clinical examination, unspecific symptoms like musculoskeletal pain, usually located in the pelvis, the shoulders or the proximal part of the muscles, can be found. Based on those symptoms, diseases with comparable symptoms such as rheumatic diseases must be excluded with the help of differential diagnosis (Reuss-Borst 2014, Rader, Corsten et al. 2015). Osteoporosis, a skeletal disease, is characterized by a decrease in bone mass and pathological changes of the microarchitecture due to a low serum level of 25(OH)D, leading to an elevated risk of osteoporotic fractures.

Figure 3. Left: Schematic structure of a healthy bone; dense and loadable bone structure. Right: Schematic structure of an osteoporotic bone; decreased bone mass and pathological changes in microarchitecture (Wintermeyer, Ihle et al. 2016).

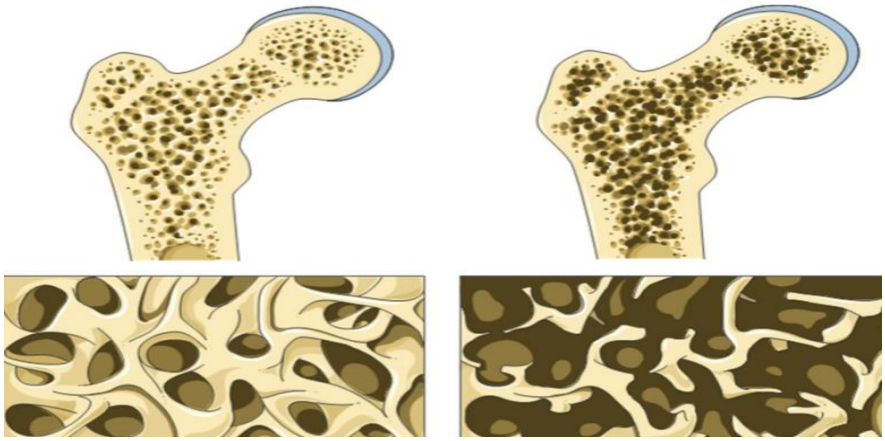

Figure 3:Schematic structure of a healthy bone

The telephone health survey by the Robert Koch Institute (RKI) in 2003 revealed that the lifetime prevalence for osteoporosis in women (aged >45) is over 14% and is even rising with increasing age . Wacker and Holick depict a prevalence of about 30% in women aged between 60 and 70 years (Wacker and Holick 2013). Per year, there are approximately 9 million osteoporosis-induced fractures globally (~2 million in the USA), among which fractures of the hip and forearm are most common (Winzenberg, van der Mei et al. 2012, Siris, Adler et al. 2014, Callegari, Reavley et al. 2015). The lifetime prevalence of an osteoporotic hip fracture among North American women is about 18% (Yoo, Moon et al. 2015). Johnell outlines a 25% increase in hip fractures between 1990 and 2000 (Johnell and Kanis 2006).

2.3 Vitamin D and osteoporosis

Osteoporosis is a common disease characterized by low bone mass and deterioration of bone microarchitecture leading to an increased risk of fracture (Gurung and Ghosh 2021). Osteoporosis related fractures are extremely common: approximately 40-50% of postmenopausal women and 25% of older men. The main role of vitamin D in bone metabolism is to increase the plasma levels of calcium and phosphorus, essential for mineralization. (Rachner, Khosla et al. 2011). All these mechanisms increase bone mineral density and reduce the risk of osteoporosis. Vitamin D in its active form (1,25(OH)2D3) is able to increase circulating levels of calcium and phosphorus to normal levels through three pathways. The first pathway is by stimulating the absorption of calcium and phosphate in the intestine, particularly in the duodenum and jejunum. This occurs due to the opening of calcium channels and by the formation of calcium-binding protein, independent of PTH

(Migliaccio, Greco et al. 2011). The second pathway, dependent on PTH, occurs through mobilization of calcium and phosphorus from bone. The increased serum PTH stimulates bone turnover, leading to bone loss. The third pathway is also dependent on the PTH stimulating the hydroxylation of 25(OH)D in the kidney to 1,25(OH)2D And involves the increase in renal retention of calcium due to increased tubular reabsorption or a decrease of filtered load (Peacock 2010). Mineralization is a passive process, but it only occurs when calcium and vitamin D are available in sufficient quantities. In vitamin D deficiency, there is a decrease in circulating levels of calcium and increased PTH levels. PTH acts by increasing P450C1 hydroxylase activity in the kidney, which consequently increases vitamin D serum levels, and is a potent agent in bone resorption. In this new phase, the circulating levels of vitamin D and calcium are normal, but the bone reserves become compromised. If vitamin D deficiency occurs for a prolonged period, substrates for synthesis of the active form of the vitamin may be reduced and the resulting bone loss can lead to osteoporosis (Holick 2006) . He also reported that among 3270 elderly French women given 1200 mg of calcium and 800 IU of vitamin D3 daily for 3 years, the risk of hip fracture was reduced by 43%, and the risk of nonvertebral fracture by 32%. A 58% reduction in nonvertebral fractures was observed in 389 men and women over the age of 65 years who were receiving 700 IU of vitamin D3 and 500 mg of calcium per day (Dawson-Hughes, Harris et al. 1997).

2.4 Vitamin D deficiency and insufficiency

Vitamin D deficiency and insufficiency The major causes of vitamin D deficiency are poor nutrition, deprivation of sunlight, consequent decline in the synthesis of cutaneous vitamin D3 and decreased renal hydroxylation of

25(OH)D by the ageing kidney (Gallagher, Riggs et al. 1979, Tsai, Heath et al. 1984). Long-lasting and severe vitamin D deficiency leads in adults to osteomalacia and in children to rickets (a bone disorder characterized by typical biochemical and bone abnormalities), along with defective mineralization, severe secondary hyperparathyroidism, hypo- calcaemia, hypophosphotaemia and an increase in total alkaline phosphatase. Vitamin D deficiency can be confirmed by measuring 25(OH)D levels which are usually very low and often undetectable. The pre- valence is high in the institutionalized and housebound elderly population (McKenna 1992).

Vitamin D insufficiency (subclinical vitamin D deficiency) is increasingly being recognized as a distinct pathological entity. In contrast to vitamin D deficiency, it is characterized by mild secondary hyperparathyroidism, normocalcaemia and normal bone mineralization. The initial fall in ionized plasma calcium stimulates parathyroid hormone secretion, which in turn stimulates renal 1a-hydroxylase and increases 1,25(OH)2D production. This restores serum calcium to the normal set-point for that individual (Brown 1991), but at the expense of increased bone turnover, and prevents the emergence of osteomalacia (Lips, Netelenbos et al. 1982, Khaw, Sneyd et al. 1992). The increase in 1,25(OH)2D in response to the parathyroid hormone stimulus in vitamin D deficiency has nevertheless been found to be inappropriate and remains within the mid–low normal laboratory refer- ence range (Chapuy, Chapuy et al. 1987, Åkesson, Lau et al. 1997). This may be partly related to the degree of substrate [25(OH)D] deficiency and possibly impaired 1a-hydroxylation of the ageing kidney, despite normal renal function (DeLuca 1997). Vitamin insufficiency is common in adults and in older people living at home. Chapuy and co-workers (Chapuy, Schott et al.

1996)found that 39% of healthy ambulatory elderly women recruited from the general community in France had a 25(OH)D level of <12 ng/ml. In a further study, they investigated the vitamin D status of a middle-aged general adult urban population (aged 45–65 years) and found that 14% had a 25(OH)D level of <12 ng/ml) (Chapuy, Preziosi et al. 1997). Untreated, vitamin D insufficiency progresses to bone loss and thus increased risk of fracture, but this is further compounded with ageing. Peak bone mass is achieved around the age of 20–30 years, followed by a period (Sahota, Masud et al. 1999)of consolidation and then an age-related decline in osteoblastic function, leading to an excess of bone resorption over formation and consequent bone loss. Peak bone mass and rate of bone loss are important in the development of osteoporosis. However, when there is vitamin D insufficiency, bone resorption is amplified, further increasing fracture risk. This pathophysio-logical process has been recognized in patients present- ing with osteoporotic hip fractures and occurs also in fit elderly people with established vertebral osteoporosis, (Sahota, Masud et al. 1999) which encompasses most osteoporotic patients presenting to doctors.

2.5 Therapeutic intervention

2.5.1 Calcium therapy

In adults, calcium supplementation reduces the rate of age-related bone loss. A review of 20 prospective studies concluded that calcium supplementation reduced bone loss on average by about 1% per year in postmenopausal women (Recker, Hinders et al. 1996). The effect of calcium in reducing the incidence of fractures has, however, been inconsistent. Recker et al. (Recker, Hinders et al. 1996) found that 1200 mg of calcium daily reduced the incidence of

vertebral fractures in women with low calcium intakes and with one or more vertebral fracture, but did not reduce the risk of the first vertebral fracture. In contrast, Chevalley et al. (Soroko, Barrett-Connor et al. 1994) observed a marked reduction in the incidence of first vertebral fracture with calcium supplementation, although all patients were vitamin D-replete.

Table 1. The relationship of the biochemical indices, bone turnover and bone mineral density in vitamin D deficiency, insufficiency and sufficiency

Table 1:The relationship of the biochemical

	Deficiency	**Insufficiency**	**Sufficiency**
25(OH)D, ng/ml	**0–5**	**5–30**	**>30**
Parathyroid hormone	**High**	**High normal**	**Normal**
1,25(OH)2D	**Low/normal**	**Low normal**	**Normal**
Bone turnover	**High**	**High normal**	**Normal**
Bone	**Osteomalacia/low bone mass**	**Low bone mass**	**Normal**

25(OH)D, 25-hydroxylated vitamin D (calcidiol); 1,25(OH)2D, 1,25-dihydroxy vitamin D.

have examined the relationship between calcium and hip fracture risk, and these have produced conflicting results (Paganini-Hill, Ross et al. 1981, Kanis, Johnell et al. 1992). No studies have evaluated the effects of calcium to reduce the risk of a second hip fracture in vitamin D-insufficient subjects.

2.5.2 Combination vitamin D and calcium

The use of combination vitamin D and calcium therapy has nevertheless shown a consistent reduction in non- vertebral fractures. Chapuy et al. (Soroko, Barrett-Connor et al. 1994) showed that supplementation with 1.2g calcium and 800 IU vitamin D3 over 18 months resulted in a 43% reduction in hip fractures and a 32% reduction in the total number of non-vertebral fractures in institutionalized, vitamin D-insufficient elderly women compared with the placebo group, with a mean reduction of 47% in secondary hyperparathyroidism. Previous osteoporotic fracture were present in some of these patients, but sub-analysis of prevalent fractures and the reduction in second fractures was not carried out. Dawson-Hughes and co-workers' study of. patients over the age of 65 years living at home showed that treatment for 3 years with 500 mg of calcium plus 700 IU of vitamin D3 increased bone mineral density at both hip and spine (Heaney, Barger-Lux et al. 1997). The reduction of non-vertebral fractures was of a similar magnitude to that in Chapuy and co- workers' study, but the absolute numbers of fractures in the study were small. In this study it was unclear what proportion of patients were vitamin D- insufficient, although there was a 33% mean reduction in parathyroid hormone). In any event, the recommended intake of 1200 mg/d of calcium for the general population should cover calcium needs. The level of vitamin D needed to increase serum 25(OH)D by given increments had been examined. One microgram (or 40 IU) will increase serum 25(OH)D by 0.6–1.1 nM. A bigger increment is observed in subjects with lower starting levels. Age does not seem to influence the increment. In Toronto, Canada, in the wintertime, an intake of

1000 IU of vitamin D3 brought the mean 25(OH)D value to near 75 nM and 4000 IU per day brought each participant's level to 75 nM or higher.

Conclusion

Vitamin D and calcium are important in the mechanical and structural integrity of the skeleton. Subclinical vitamin D deficiency (vitamin D insufficiency) is common in the fit, active elderly population and leads to an amplification of age-related bone turnover, bone loss and thus increased risk of fracture, mediated by secondary hyperparathyroidism. Daily supplementation with vitamin D can reduce the secondary hyperparathyroidism and increase bone marrow den- sity but only combination calcium and vitamin D therapy has been shown to be effective in reducing non-vertebral fractures.

RECOMMENDATIONS

My recommendation to citizens, especially those who have problems with vitamin D deficiency. Try to devise a suitable program for eating foods that are rich in vitamin D. Vitamin D levels are high in fish ,cheese, milk, grains, eggs.

Also, they allow to be hit by sun more than needed to increase vitamin D in the skin. On the condition that it does not cause superfluous. In some situations, the person can use many kinds of medicines to increase the level of vitamin D in them, but it is better for the patient to use natural methods more. Only in some situations can he take medicine.

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پوختە

ڤیتامین D بە بری پێویست زۆر گرنگە بۆ تەندروستی ئیسکەکان ڤیتامین هانی مژینی کالیسیۆم ، فۆسفۆر دەدات لە ریخۆلە باریکە ،دەبێتە هۆی زیادبوونی ئاستی کالیسۆم و فۆسفۆر ، لە زیندەچالاکی ئسکدا ڤیتامین D هەلدەستی بە رێکخستنی چالاکی .
رێگری دەکات بە زۆر دەردانی پاراثایرۆید هۆرمۆن و وە دەبێتە هۆی پێش بردنی دروست بونی ئێسك و رێگری دەکات لە فەشەل بونی ئێسکەکان هەروەها چارەسەری فەشەل بونی ئێسکەکان دەکات وە ئەو بەلگەیە پشتگیری دەکرێت لە لایەن زۆرێك لە خوێندنەوەو کیلینیکی یەکان کاریگەریەکانی کەمی ڤیتامین D دەبێتە هۆی زۆر بونی هۆرمۆنی ثایرۆیرۆید و کەم بونەوەی ئێسکەکان کە دەبێتە هۆی فەشەل بونی ئێسك و شکانی ئێسکەکان باوترین سەرچاوەی ڤیتامین D بۆ منداڵ و هەرزەکاران بریتیە لە خۆدانە بەر خۆر کە فۆتۆنەکانی تیشکی سەرو وەنەوشەی B هەڵئەمژرێت لە لایەن هایدرۆکۆلیسترۆل کە لە پێست دا هەن کە دەبێتە هۆی گواستنەوەی بۆ ڤیتامین D ڤیتامین D لە جگەر زیندە چالاکی بەسەر دادێت دەگۆڕێت بۆ 25 هایدرۆکسی ڤیتامین D کە دواتر لە گورچیلە دەگۆڕێت بۆ شێوەی بەکارهێنانی - دژە خۆر و جل و بەرگو تەمەن - بەشێوەیەکی بەرچاو کاریگەری لەسەر دروست بونی ڤیتامین D هەیە بەشێوەیەکی سروشتی بڕێکی کەم لە خواردنەکان ڤیتامین D یان تێدایە.

زانکۆی سەلاحەدین-هەولێر

Salahaddin University – Erbil

رۆلی ڤیتامین D لە ئیسکە نەرمە

پرۆژەی دەرچوون کە دەدرێتە لێژنەی کۆلێژی پەروەردەی شەقلاوە زانکۆی سەلاحەدین وەک بەشێک لە بەدیهینانی داواکاری بۆ بروانامەی بەکالۆریۆسی پەروەردەی زانستی بایۆلوژی

ئامادەکراوە لە لایەن:

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