Vitamin D and Rehabilitation: Improving Functional Outcomes

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Invited Review

Vitamin D and Rehabilitation: Improving Functional Outcomes

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ABSTRACT: Vitamin D inadequacy is pandemic among rehabilitation patients in both inpatient and outpatient settings. Male and female patients of all ages and ethnic backgrounds are affected. Vitamin D deficiency causes osteopenia, precipitates and exacerbates osteoporosis, causes the painful bone disease osteomalacia, and worsens proximal muscle strength and postural sway. Vitamin D inadequacy can be prevented by sensible sun exposure and adequate dietary intake with supplementation. Vitamin D status is determined by measurement of serum 25-hydroxyvitamin D. The recommended healthful serum level is between 30 and 60 ng/mL. 25-Hydroxyvitamin D levels of >30 ng/mL are sufficient to suppress parathyroid hormone production and to maximize the efficiency of dietary calcium absorption from the small intestine. This can be accomplished by ingesting 1000 IU of vitamin D_3 per day, or by taking 50,000 IU of vitamin D_2 every 2 weeks. Vitamin D toxicity is observed when 25-hydroxyvitamin D levels exceed 150 ng/mL. Identification and treatment of vitamin D deficiency reduces the risk of vertebral and nonvertebral fractures by improving bone health and musculoskeletal function. Vitamin D deficiency and osteomalacia should be considered in the differential diagnosis of patients with musculoskeletal pain, fibromyalgia, chronic fatigue syndrome, or myositis. There is a need for better education of health professionals and the general public regarding the optimization of vitamin D status in the care of rehabilitation patients.

Despite medical advances of the 20th century, vitamin D deficiency is still pandemic.[1] Close to 1 billion people worldwide have vitamin D deficiency. Classically, vitamin D deficiency is associated with rickets in children. In adults, vitamin D deficiency is manifested as osteomalacia, a painful condition of defective skeletal mineralization, and as the painless condition of osteoporosis that causes skeletal fragility and fractures.[1,2]

Vitamin D also plays an important role in regulation of proliferation and differentiation in a variety of cells and tissues not associated with calcium metabolism.[1–4] Receptors for vitamin D (VDR) were found in a variety of body tissues and cells, including brain, heart, breast, prostate, gonads, colon, pancreas, monocytes, and activated T and B lymphocytes.[3–6] Vitamin D is important in maintaining muscle strength through its action on VDR in muscle tissue.[7]

Rehabilitation patients in both inpatient and outpatient settings are a high-risk population prone to develop and have the consequences of vitamin D deficiency.[8,9] The functional outcomes of patients in rehabilitation programs depend on correct diagnosis and appropriate treatment of those affected. The goal of this review is to update the readers on the current concepts in vitamin D physiology and clinical applications as they relate to patients in rehabilitation settings.

Vitamin D Physiology and Metabolism

The main source of vitamin D is cutaneous synthesis from the cholesterol precursor 7-dehydrocholesterol (7-DHC, provitamin D_3).[1,4] 7-DHC absorbs solar energies between 290 and 315 nm (ultraviolet B [UVB]) to form previtamin D_3 that undergoes rapid isomerization in the skin to vitamin D_3 (Figure 1).[10]

Vitamin D can also be obtained from diet. It comes in 2 forms: vitamin D_2 (ergocalciferol) is derived from UVB-irradiated yeast and plants, and vitamin D_3 (cholecalciferol) is found in cod liver oil and in fatty fish, such as salmon and mackerel.[2,4,10] Because natural dietary sources of vitamin D are scarce, some foods are fortified with vitamin D. In the United States, some cereals, bread products, yogurts (100 IU/serving), orange juice (100 IU/8 oz), and milk (100 IU/8 oz) are fortified with vitamin D.[1,10,11,12] Both vitamin D_2 and vitamin D_3 are used for fortification of foods and supplements. Vitamin D_2 is only about 30% as effective as vitamin D_3 in maintaining vitamin D status.[13] Thus, 3 times as much vitamin D_2 as vitamin D_3 is needed to have the same effect on a person's vitamin D status.

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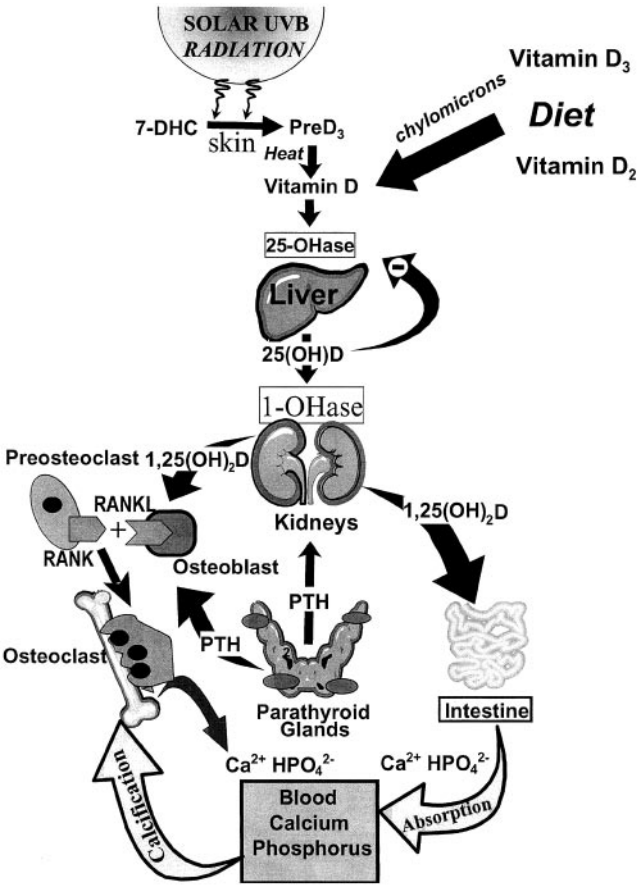

Figure 1. Schematic representation of the synthesis and metabolism of vitamin D for the purpose of regulating calcium, phosphorus, and bone metabolism. During exposure to sunlight, 7-dehydrocholesterol (7-DHC) is converted to previtamin D_3 ($preD_3$). Once formed, it immediately converts in the skin by a heat-dependent process to vitamin D_3. Excessive exposure to sunlight degrades $preD_3$ and vitamin D_3 into inactive photoproducts. Vitamins D_2 and D_3 from dietary sources is incorporated into chylomicrons, transported by the lymphatic system into the venous circulation. Vitamin D (D represents D_2 or D_3) made in the skin or ingested in the diet can be stored in fat cells. Vitamin D in the circulation is bound to the vitamin D binding protein (DBP), which transports it to the liver, where vitamin D is converted by the vitamin D-25-hydroxylase (25 OHase) to 25-hydroxyvitamin D [25(OH)D]. This is the major circulating form of vitamin D used by clinicians to measure vitamin D status. It is biologically inactive and must be converted in the kidneys by the 25-hydroxyvitamin D-1α-hydroxylase (1-OHase) to its biologically active form, 1,25-dihydroxyvitamin D [$1,25(OH)_2D$]. Various factors, including serum phosphorus (P_i) and calcium (Ca), can either increase (+) or decrease (−) the renal production of $1,25(OH)_2D$. Also, feedback regulates its own synthesis and decreases the synthesis and secretion of parathyroid hormone (PTH) in the parathyroid glands. $1,25(OH)_2D$ also increases the expression of the 25-hydroxyvitamin D-24-hydroxylase (24-OHase) to the water-soluble biologically inactive metabolite calcitroic acid, which is excreted into the urine. $1,25(OH)_2D$ enhances intestinal calcium and phosphorus absorption in the small intestine by stimulating the expression of the epithelial calcium channel (ECaC) and the calbindin 9K (calcium binding protein; CaBP). $1,25(OH)_2D$ is also recognized by its receptor in osteoblasts, causing an increase in the expression of receptor activator of NFκB ligand (RANKL). Its receptor on the RANK of the preosteoclast binds RANKL, which induces the preosteoclast to become a mature osteoclast. The mature osteoclast removes calcium and phosphorus from the bone to maintain blood calcium and phosphorus levels. Adequate calcium and phosphorus levels promote the mineralization of the skeleton. Reproduced with permission of Michael F. Holick. Copyright 2006.

Once vitamins D_2 or D_3 enters the circulation from either cutaneous synthesis or from diet, it binds to vitamin D binding protein (DBP).[1,2] It is then transported to the liver, where it undergoes first hydroxylation to 25-hydroxyvitamin D [25(OH)D; Figure 1]. 25(OH)D is biologically inert and must undergo second hydroxylation in the kidney to become the biologically active 1,25-dihydroxyvitamin D [$1,25(OH)_2D$].

The major biologic function of $1,25(OH)_2D$ is to maintain the serum calcium concentration in physiologic range to maximize metabolic and neuromuscular activity and to maintain calcium and phosphorus in the normal range for bone mineralization.[1–4] It accomplishes this by acting on the VDR in the small intestine to increase calcium and phosphorus absorption. The efficiency of the intestinal calcium and phosphorus absorption in vitamin D deficiency is ~10%–15% for dietary calcium and ~60% for dietary phosphorus.[1,14] Vitamin D sufficiency enhances dietary calcium absorption to ~30%–40% and dietary phosphorus absorption to ~70%–80%. During pregnancy and lactation, the $1,25(OH)_2D$ production is increased, which raises calcium absorption up to ~60%–80%.[4]

Renal production of $1,25(OH)_2D$ is tightly regulated by parathyroid hormone (PTH), serum calcium and phosphorus.[2,4] In a vitamin D–deficient state, there is a decline in the efficiency of intestinal calcium absorption and serum ionized calcium concentration. This decline is detected by calcium sensor in the parathyroid glands, resulting in the increased production and secretion of PTH. In turn, PTH acts on the kidneys to increase production of $1,25(OH)_2D$. In addition, hypocalcemia and hypophosphatemia are potent stimulators of $1,25(OH)_2D$ production.

Both PTH and $1,25(OH)_2D$ act on osteoblasts to induce expression of receptor activator of NFκB ligand (RANKL).[4,6,15] The preosteoclasts carry the receptor activator of NFκB (RANK). When dietary calcium intake is inadequate or in a vitamin D–deficient state, the interaction of RANKL on osteoblasts with RANK on preosteoclasts induces osteoclastogenesis and enhances mobilization of calcium from the skeleton into the circulation.

Noncalcemic Functions of Vitamin D

$1,25(OH)_2D$ plays an important role in regulation of cell growth, immune function, blood pressure control, and insulin production.[1–4,6] The antiproliferative and prodifferentiating properties of $1,25(OH)_2D$ and its analogs have been successfully developed to treat the hyperproliferative skin disease psoriasis and are in development to treat prostate, breast, liver, and colon cancer.[4,16] $1,25(OH)_2D$ suppresses renin production in the kidneys and stimulates insulin production and secretion by the β-islet cells.[17] Activated T and B lymphocytes, monocytes, and macrophages all respond to $1,25(OH)_2D$, resulting in modulation of immune function and reducing risk of infectious disease, including tuberculosis and possibly influenza.[1,4,6]

Vitamin D improves musculoskeletal function by exerting direct effect on muscle tissue, which has a VDR.[7,18,19] Supplementation with vitamin D leads to an increase in the cross-sectional area of fast-twitch-type IIA fibers, improvement in proximal muscle strength and postural body sway, and decrease in the number of falls.[20–23]

Definition of Vitamin D Deficiency

The best assay to determine vitamin D status is to measure serum 25(OH)D.[1–4,10] In contrast, the level of $1,25(OH)_2D$ is not a reliable indicator of vitamin D status and often is misleading. $1,25(OH)_2D$ has a much shorter half-life, only 4–6 hours, as compared with 2 weeks for 25(OH)D, and concentration 1000 times less than 25(OH)D.[4] As levels of 25(OH)D fall below 20 ng/mL, there is still enough substrate to produce $1,25(OH)_2D$. This is why in a vitamin D–deficient state, the levels of $1,25(OH)_2D$ may be normal or even elevated. Nonetheless, measuring $1,25(OH)_2D$ is useful for the differential diagnosis of hypercalcemia and may be helpful for evaluation of metabolic bone disease in patients with severe renal disease.

The normal range of 25(OH)D at 10–55 ng/mL was established in the past by sampling what was assumed to be vitamin D–sufficient population and determining the mean and variation of 2 standard deviations (SDs).[1,2,24] However, the unexpected prevalence of vitamin D deficiency in the general population of all ages, ethnic groups, and both genders led to significant underestimation of the normal range.[2,24] Provocative interventions revealed that giving 50,000 IU of vitamin D_2 once a week for 8 weeks caused a reduction in PTH levels in patients with baseline levels of 25(OH)D <20 ng/mL and no significant change above 20 ng/mL. Studies plotted serum PTH levels as a function of 25(OH)D, and a study of 25(OH)D levels in relation to calcium absorption was reported as a more physiologic method to determine the lower limit of normal range for 25(OH)D. PTH was maximally suppressed when 25(OH)D levels were above 30 ng/mL.[1,24–29] Intestinal calcium absorption was maximized at 25(OH)D levels >32 ng/mL.[14] Thus, most of the experts now agree that a 25(OH)D of <20 ng/mL is vitamin D deficiency and levels between 21 and 29 ng/mL are vitamin D insufficiency.[2]

Risk Factors for Vitamin D Deficiency

Factors that interfere with vitamin D intake, absorption, synthesis, or metabolism result in vitamin D deficiency.[1,3] Advanced age, non-Caucasian ethnicity, inadequate exposure to solar radiation, poor dietary intake, obesity, and medications that impair vitamin D activation or accelerate clearance are known risk factors for vitamin D deficiency.

The cutaneous synthesis of vitamin D in the elderly is markedly reduced due to a decrease in 7-DHC concentrations in the epidermis.[30,31] Exposure to the same amount of sunlight in a person >70 years old results in <30% production of vitamin D_3 as compared with a young adult. Both melanin and sunscreens compete with 7-DHC for UVB photons. People of dark skin color require 5–10 times longer exposure to sunlight to make the same amount of vitamin D_3 as their white counterparts.[32–34] Similarly, a sunscreen with skin protecting factor of 8 (SPF8) reduces cutaneous production of vitamin D_3 by 95%.[35]

The amount of UVB radiation that reaches the Earth's surface depends on the zenith angle of the sun. In the northern latitudes, winter months, and early mornings and late afternoons during summer, the angle is so oblique that most of the UVB photons are absorbed by ozone in the atmosphere, preventing cutaneous synthesis of vitamin D_3. At latitude of 42° north (Boston, MA), the sunlight is insufficient to produce vitamin D_3 between the months of November through February.[36] In comparison, at latitude of 52° north (Edmonton, Canada), this period is extended to include October through March.[36] The amount of UVB exposure can also be influenced by cultural and religious practices of wearing protective clothing and avoiding direct sunlight, by participating in indoor activities during daytime, or in patients with limited functional mobility.[1,10,37]

Fortified milk is the major dietary source of vitamin D_2 or vitamin D_3. However, the vitamin D content is highly variable, with <50% of milk sampled having at least 50% of the vitamin D content identified on the label, and ~20% of skim milk having no detectable vitamin D.[12] Fatty fish such as salmon and mackerel are a natural source of vitamin D, though farmed salmon has only 10%–25% of the vitamin D content compared with wild salmon.[38] Dietary preferences or the higher prevalence of lactose intolerance with age, especially in African American populations, can lead to inadequate dietary intake of both calcium and vitamin D. Patients with fat malabsorption syndromes, such as cystic fibrosis and Crohn's disease, are at especially high risk to develop vitamin D deficiency.[39] Obesity

is an independent risk factor due to sequestration of vitamin D in the adipose tissue.[40]

Many medications, including antiseizure medications, glucocorticoids, rifampin, and over-the-counter products such as St John's wort, enhance the catabolism of vitamin D through the action of pregnane-X-receptor (PXR).[4,41–43] When PXR binds to one of these drugs and becomes activated, it complexes with retinoic acid X receptor (RXR). PXR-RXR drug complex binds to the VDR responsive element of the 25(OH)D-24-hydroxylase and enhances its expression, resulting in the degradation of 25(OH)D and $1,25(OH)_2D$.

Prevalence of Vitamin D Deficiency

Vitamin D deficiency remains a major health problem in all age groups. Sullivan et al[44] reported that 48% of white girls 9–11 years of age in Maine were vitamin D deficient (25(OH)D <20 ng/mL) at the end of winter, and 17% of them remained vitamin D deficient at the end of summer. In Boston, 42% of adolescents and 32% of young adults were found to be vitamin D deficient.[1,45] The National Health and Nutrition Examination Survey (NHANES) III revealed that 42% of African American women 15–49 years old had severe vitamin D deficiency (25(OH)D <15 ng/mL) at the end of winter.[46] Approximately 50% of community-living elderly in Boston and Baltimore were found to be vitamin D deficient throughout the year.[4] The rate of vitamin D deficiency is even higher in hospitalized or institutionalized individuals. Thomas et al[47] reported that 57% of medical inpatients were vitamin D deficient.

Vitamin D deficiency is highly prevalent in rehabilitation inpatients and outpatients. In a recent study of inpatients admitted to subacute rehabilitation facility in Boston, 49.1% were found to be vitamin D deficient.[8] Vitamin D deficiency was equally prevalent in both male (46.7%) and female (52.2%) patients of all ethnic backgrounds (Figure 2). Plotnikoff and Quigley[48] reported that 93% of outpatients presenting with musculoskeletal pain syndromes in Minneapolis were vitamin D deficient.

Vitamin D Deficiency, Bone Mineral Density, and Fracture Risk

Maintaining adequate calcium and vitamin D status is essential for bone health. A transient decrease in serum ionized calcium concentration due to either inadequate dietary calcium intake or secondary to decrease in calcium absorption from the small intestine as a result of vitamin D deficiency leads to secondary hyperthyroidism and increase in bone resorption. A long-term effect is the decrease in bone mineral density and increased fracture risk (Figure 3).[1–4]

The NHANES III survey of 13,432 noninstitutional residents of diverse age and ethnic background in the United States demonstrated strong positive correlation between 25(OH)D and total hip bone mineral density.[49] Other studies also reported positive correlation between 25(OH)D and bone mineral density in the lumbar spine, femoral neck, and forearm.[50–52]

Vitamin D and calcium supplementation was shown to moderately reduce bone loss and to significantly reduce the risk of nonvertebral fractures. In

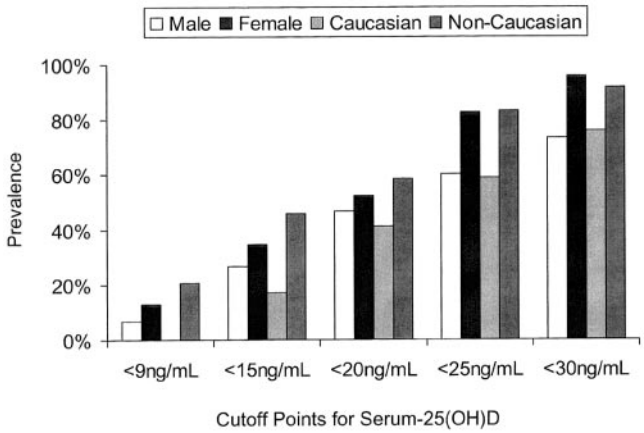

Figure 2. Prevalence of Vitamin D inadequacy in rehabilitation inpatients; percentage of rehabilitation inpatients with serum 25-hydroxyvitamin D [25(OH)D] concentrations below predefined cutoffs of <9, <15, <20, <25, and <30 ng/mL. Total of 53 patients; the mean age was 60.2 ± 14.1, 30 men (56.6%), 23 women (43.4%), 29 Caucasian (54.7%), 20 African American (37.7%), 2 Hispanic (3.7%), and 1 Asian (1.9%). Reproduced with permission of Michael F. Holick. Copyright 2006.

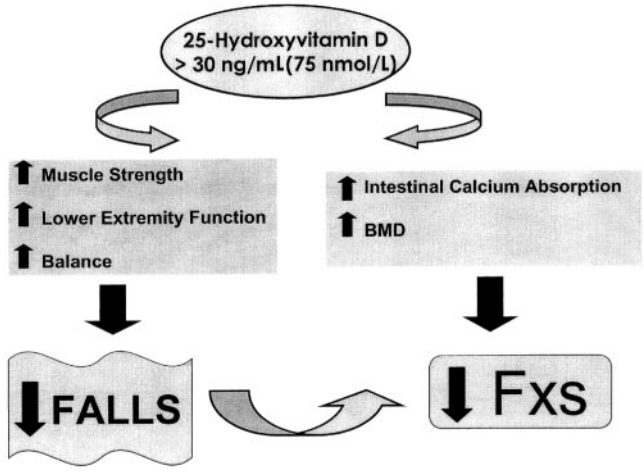

Figure 3. The effect of improving vitamin D status. Maintaining serum 25-hydroxyvitamin D level above 30 ng/mL will improve muscle strength, lower-extremity function, balance; it will also increase intestinal calcium absorption and bone mineral density and therefore will result in reduction in the risk of falls and fracture. Reproduced with permission of Michael F. Holick. Copyright 2006. BMD, bone mineral density; Fxs, fractures.

a randomized, placebo-control study, Dawson-Hughes et al[53] evaluated the effect of daily supplementation with 700 IU of vitamin D_3 and 500 mg of elemental calcium on bone mineral density and the incidence of nonvertebral fractures in 176 men and 213 women 65 years of age or older living in the community. They determined that over a period of 3 years, there was significant positive effect on the change in bone mineral density at the total body, femoral neck, and lumbar spine, with the cumulative incidence of a first fracture decreased to 5.9% in the calcium–vitamin D group as compared with 12.9% in the placebo group. Chapuy et al[54] demonstrated that daily supplementation with 800 IU of vitamin D_3 and 1200 mg of calcium in 3270 healthy ambulatory elderly women over 18 months decreased the risk of hip fractures by 43% and other nonvertebral fractures by 32%.

Three recent intervention trials challenge the findings of decreased risk of fracture with vitamin D and calcium supplementation. Porthouse et al[55] studied the effect of daily oral supplementation with 800 IU of vitamin D_3 and 1000 mg of calcium over a period of 25 months in 3314 women aged 70 years and older with 1 or more risk factors for hip fractures. In the RECORD trial, Grant et al[56] examined 5292 people age 70 years and older who had previous history of fragility fracture. Patients were randomly assigned to 800 IU of vitamin D_3 and 1000 mg of calcium, vitamin D_3 alone, or placebo and were followed up at 2–5 years. In the Women's Health Initiative (WHI) study, 36,282 postmenopausal women 50–79 years of age were randomly assigned to receive 1000 mg of calcium with 400 IU of vitamin D_3 or placebo.[57] Patients had an average follow-up of 7 years. All 3 studies failed to show significant reduction in the risk of fracture in patients treated with vitamin D_3. However, the compliance was a major problem in all studies, with $<60\%$ adhering to the regimen. In the WHI study, the amount of vitamin D_3 supplementation was only 40% of what is now recognized as the minimum adequate vitamin D intake. Both studies are illustrative of the fact that the reduction in fracture risk cannot be achieved with suboptimal supplementation and poor patient compliance.

Vitamin D and Muscle Strength

Decrease in the fracture risk with vitamin D supplementation may not only be mediated by reduction in bone loss but also by direct effect of vitamin D on skeletal muscle.[18,19] There is a strong association between higher serum 25(OH)D concentrations and muscle strength, physical activity, and reduced falls in the elderly.[7,20–23,58–60] In a study of 246 hospitalized and 103 community living elderly, Mowe et al[58] demonstrated positive correlation between 25(OH)D and arm muscle strength, ability to climb stairs, physical activity, and the absence of falls. Dhesi et al[59] reported that vitamin D deficiency correlated with impaired functional performance, psychomotor function, and muscle strength in elderly people prone to falls. In a study of 237 postmenopausal women with osteoporosis, Pfeifer et al[21] reported a strong correlation between 25(OH)D and increased body sway and elevated risk of falls.

Bischoff et al[22] demonstrated in a randomized, controlled trial that daily supplementation with 800 IU of vitamin D_3 and 1200 mg of calcium improved musculoskeletal function (knee flexor and extensor strength, grip strength, and timed up-and-go test) and reduced the risk of falls by 49% in institutionalized elderly. Two recent meta-analysis studies examined the effect of vitamin D supplementation on falls and fractures in ambulatory or institutionalized elderly. The first meta-analysis of 5 double-blind randomized controlled trials (RCTs) revealed that vitamin D supplementation lowered the risk of falling by 22%.[23] In the second meta-analysis of 12 double-blind RCTs, the relative risk reduction of 26% in hip fractures and 23% in nonvertebral fractures was reported, with oral supplementation of at least 700–800 IU needed to achieve the benefit.[60]

Vitamin D and Pain Syndromes

There is increasing evidence that vitamin D deficiency is associated with musculoskeletal pain in both children and adults. Plotnikoff and Quigley[48] reported that 93% of adults and children who presented with persistent musculoskeletal pain who did not meet strict criteria for fibromyalgia defined by the American College of Rheumatology were vitamin D deficient. In a study of Danish women of Arab descent who presented with muscle pains and weakness, 88% were found to be severely vitamin D deficient.[61] Erkal et al[37] observed a strong correlation between low 25(OH)D and higher prevalence and longer duration of generalized bone or muscle pain often diagnosed as fibromyalgia among Turkish women living in Germany. Gloth et al[62] reported 5 cases of treatment-resistant musculoskeletal pain that completely resolved in 5–7 days after supplementation with vitamin D. Similarly, Malabanan et al[63] showed marked improvement in bone mineral density and resolution of musculoskeletal symptoms with correction of vitamin D deficiency.

Osteomalacia is a known cause of persistent nonspecific musculoskeletal pain.[2,64,65] The pain, typically described as aching and throbbing, can be elicited on physical examination by applying minimal pressure over sternum or anterior tibia.[2] This is thought to be due to poorly mineralized osteoid that acts like gelatin underneath the periosteum. It becomes hydrated, pushing outward on the richly innervated periosteum, giving the sensation of throbbing, aching bone pain. In addition, the jelly-like collagen matrix provides minimal structural support, and any pressure applied to the periosteum results in deformation of the periosteal lining, eliciting "wincing"-like pain. Patients with osteomala-

cia are often misdiagnosed as having fibromyalgia, chronic fatigue syndrome, or myositis.[2,48,64,65]

Prevention and Treatment of Vitamin D Deficiency

Vitamin D deficiency can be prevented through sensible sun exposure, adequate dietary and supplemental intake, or pharmacologic vitamin D treatment. Children and adults can expose arms and legs to sunlight 2–3 times a week for about 5–10 minutes, depending on time of the day, season, and latitude, before applying a sunscreen.[1,2,36,66] Recommendations for vitamin D and calcium intakes were developed by the Institute of Medicine (IOM), with the most recent set of guidelines published in 1997.[67] The IOM-recommended calcium intake is 500 mg/d for ages 1–3 years, 800 mg/d for ages 4–8 years, 1300 mg/d for ages 9–18, 1000 mg/d for ages 19–50, and 1200 mg/d for 51 years and older. Calcium absorption is more efficient in single doses of $\leq$500 mg.[68]

Fractional calcium absorption from calcium carbonate, the most commonly used supplement, is significantly better when taken with a meal. Calcium citrate might be a better alternative in patients with achlorhydria or a history of kidney stones or renal disease.[69]

The vitamin D daily intake recommendations from the IOM are as follows: 200 IU of vitamin D for all children and adults up to 50 years old, 400 IU for adults 51–70 years old, 600 IU for adults >71 years old. However, recent data showed that approximately 1000 IU of vitamin D_3 daily is needed to maintain serum 25(OH)D concentration above 30 ng/mL.[1,14,70,71] Care must be taken to insure adequate vitamin D supplementation because recent studies demonstrated a high prevalence of vitamin D deficiency even in patients receiving pharmacologic therapy to treat osteoporosis while taking a multivitamin that contained vitamin D.[26]

Vitamin D deficiency is treated by administration of vitamin D_2 50,000 IU once a week for 8 weeks to fill vitamin D reservoirs in adipose tissue, followed by supplementation with 50,000 IU of vitamin D_2 twice a month.[1,72] Vitamin D toxicity usually develops when 25(OH)D is above 150 ng/mL.[73,74] A person must ingest >10,000 IU of vitamin D daily for 6 months to achieve this level. Chuck et al[75] reported that installation of UVB-emitting lights in the ceiling of an activity room was effective in maintaining serum 25(OH)D levels in nursing home patients at a lower cost than that of using oral vitamin D supplementation.

Conclusions

Vitamin D is essential for health and well-being. There is an unacceptably high level of vitamin D deficiency in men and women of all ages and ethnic backgrounds participating in inpatient and outpatient rehabilitation programs. The consequence of vitamin D deficiency is the painful bone disease osteomalacia, worsening of proximal muscle strength and postural sway, osteopenia, osteoporosis, and increased fracture risk. There is growing evidence of vitamin D's role in regulation of cell growth, immune function, blood pressure control, and insulin production, yet little has been done to adequately address vitamin D deficiency in this vulnerable group of patients. Measurement of 25(OH)D to check vitamin D status and adequate treatment of vitamin D deficiency are paramount in improving bone health and musculoskeletal function to decrease fracture risk. Vigilance for vitamin D deficiency is required by health professionals involved in care for patients with nonspecific pain syndromes.

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