CASE REPORT

Treatment of Vitamin D Deficiency Due to Crohn's Disease With Tanning Bed Ultraviolet B Radiation

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In Crohn's disease, severe skeletal demineralization, secondary hyperparathyroidism, and muscle weakness can occur. This may be caused by impaired vitamin D absorption, resulting from extensive intestinal disease and resection of duodenum and jejunum, where vitamin D is absorbed. We report a 57-year-old woman with a long history of Crohn's disease and short-bowel syndrome who had only 2 feet of small intestine remaining after 3 bowel resections. She was taking a daily multivitamin containing 400 IU of vitamin D_3 and was dependent on total parenteral nutrition that contained 200 IU of vitamin D and calcium (18 mEq in a 1-L bag infused over 8 hours daily) for a period of 36 months. Despite the above replacement, she complained of bone pain and muscle weakness, and she continued to be vitamin D-deficient with a 25(OH)D level <20 ng/mL. She was then exposed to ultraviolet B (UVB) radiation in a tanning bed wearing a 1-piece bathing suit for 10 minutes, 3 times a week for 6 months at the General Clinical Research Center, Boston University Medical Center. She tolerated the irradiation well without evidence of erythema. After 4 weeks, her serum 25(OH)D level increased by 357% from 7 to 32 ng/mL, parathyroid hormone level decreased by 52% from 92 to 44 pg/mL, and the serum calcium level increased from 7.8 to 8.5 mg/dL. After 6 months of UVB treatment, her serum 25(OH)D level was maintained in the normal range and was free of muscle weakness, and bone and muscle pain.

Vitamin D is a seco-steroid hormone essential for maintenance of a healthy skeleton throughout our lives.[1,2] The main function of vitamin D is to preserve calcium and phosphorus homeostasis by increasing the efficiency of intestinal calcium and phosphorus absorption to maintain signal transduction, metabolic activities, neuromuscular function, and to promote skeletal mineralization.[3] In the early years of the last century, before the identification of vitamin D, Wisner and Whipple[4,5] reported that fat malabsorption secondary to biliary obstruction led to osteoporosis. In Crohn's disease, when there is impaired absorption of vitamin D because of duodenum and jejunum resection where vitamin D is absorbed, severe skeletal demineralization, secondary hyperparathyroidism, and muscle weakness can occur.[6–8] Decreased muscle strength among vitamin D-deficient individuals indicates that myopathy precedes bone disease.[9]

In this report, we show that a tanning bed that emits levels of ultraviolet B (UVB) radiation that mimics sunlight significantly increased the circulating concentration of 25-hydroxyvitaminD [25(OH)D], and decreased parathyroid hormone (PTH) and bone pain in a patient with severe intestinal fat malabsorption caused by Crohn's disease and multiple small-bowel resections who was suffering with unrelenting bone pain and muscle weakness.

Case Report

A 57-year-old woman with a long history of Crohn's disease and short-bowel syndrome had only 2 feet of small intestine remaining after 3 bowel resections, which caused severe fat malabsorption (>14 g of fat in stool daily) and diarrhea. At age 50 (after menopause), she had been on hormone-replacement therapy with conjugated estrogen (Premarin, Wyeth Ayerst Pharmaceuticals, Philadelphia, PA) 0.625 mg by mouth daily to prevent osteoporosis, and her height had not changed.

To support her nutritional status, the patient was dependent on total parenteral nutrition (TPN) which contained 200 IU of vitamin D and calcium (18 mEq in 1-L bag infused over 8 hours daily). The patient avoided milk products because of bloating and diarrhea. The only dietary vitamin D intake was available from a multivi-

Abbreviations used in this paper: 25(OH)D, 25-hydroxyvitaminD; MED, minimal erythema dose; PTH, parathyroid hormone; TPN, total parenteral nutrition; UVB, ultraviolet B.

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tamin containing 400 IU of vitamin D, which she was taking daily, and occasionally milk products. Despite the above replacement with vitamin D and 1000 mg of calcium for 3 years, she continued to have vitamin D deficiency, with 25(OH) D levels persistently low (<20 ng/mL). She was given oral replacement of vitamin D_2 (50,000 U once a week for 8 weeks) but she was unable to tolerate the vitamin D because of dyspepsia. She had a vitamin D absorption test that showed a 50% to 70% reduction in absorption of vitamin D.[5]

The patient complained of diffuse muscle pain, weakness, and severe unrelenting sternal, arm, and leg aching. A dual-energy radiograph absorptiometry scan (Norland XR-26 Fort Atkinson, WI) was obtained and revealed osteopenia (T-score, −1.98) of the lumbar spine and osteoporosis of the hip (T-score, −3.70). At that time, her serum 25(OH)D level was 7 ng/mL (normal range, 15–45 ng/mL), PTH was 92 pg/mL, (normal range, PTH 10–45 pg/mL), and calcium was 7.8 mg/dL (normal range, Ca 8.4–10.2 mg/dL), and albumin 3.8 mg/dL (normal range, 3.5–4.4 mg/dL) all performed at Nichols Institute (San Juan Capistrano, CA).

We exposed the patient to radiation from a tanning bed equipped with Medisun lamps (Wolff System Technology Corporation, Atlanta, GA) that contained UVB radiation (290–315 nm) as previously described.[10] The patient did not have any sun exposure before being treated with the tanning bed radiation because of her debilitating disease. She was exposed to UVB radiation (via a tanning bed) in a 1-piece bathing suit for 10 minutes, which is equivalent to 0.75 minimal erythema dose (MED) radiation for her type II skin, 3 times a week for 6 months at General Clinical Research Center at Boston University Medical Center. She tolerated the irradiation well, without evidence of erythema and maintaining her 25(OH)D levels in the normal range (>20 ng/mL). After 4 weeks, her serum 25(OH)D level increased by 357% to 32 ng/mL, PTH level decreased by 52% to 44 pg/mL, and the serum calcium level increased to 8.5 mg/dL. The albumin remained the same during the treatment period. After 6 months of exposure to a tanning bed 3 times a week, her serum 25(OH)D level was 27 ng/mL, PTH was 17 pg/mL, serum calcium was 10.1 mg/dL (Figure 1), and the patient was free of muscle weakness, and bone and muscle pain. The patient's vitamin D status declined and diffuse muscle and bone pain reappeared after she stopped the exposure to UVB radiation because of frequent admissions to the hospital secondary to line sepsis, electrolyte abnormalities, and liver failure.

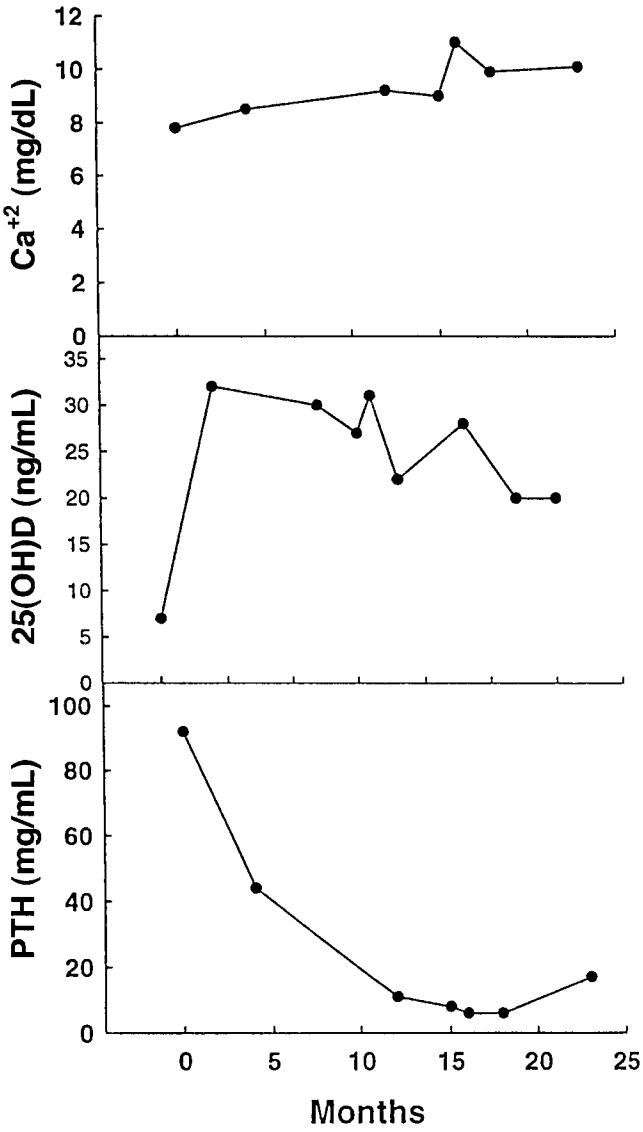

Figure 1. Serum 25(OH)D, PTH, and calcium levels in a patient with Crohn's disease who had whole-body UVB exposure for 10 minutes, 3 times a week for 6 months.

Discussion

In patients with intestinal disease, resection, or bypass there is impaired absorption of both vitamin D and calcium. The degree of malabsorption of vitamin D and calcium is variable and, in general, depends on the severity of intestinal malabsorption.[5,6] Even with close surveillance, patients with short-bowel syndrome and malabsorption are prone to vitamin D deficiency and nutritional osteomalacia.[6] In Crohn's disease, vitamin D depletion is uncommon in the absence of intestinal resection or concomitant cholestyramine therapy, because vitamin D is absorbed in the duodenum and jejunum, which is usually unaffected by Crohn's disease.[6] The causes of vitamin D deficiency are multifactorial, and the onset is insidious. Gastrointestinal dysfunction and fat-

malabsorption syndromes are often associated with vitamin D deficiency because of inadequate amounts of vitamin D entering the system, from either limited dietary intake or reduced cutaneous production by sun exposure and malabsorption of oral vitamin D.[11]

Patients with malabsorption also tend to avoid dairy products because of concominant lactose intolerance and fear of worsening diarrhea. A high index of suspicion should be maintained in these patients, with frequent follow-up and nutrition assessment, including a serum 25(OH)D level.[11] Individuals may not respond to intravenous replacement of vitamin D because the amount that it contains (400 IU) may not be enough to treat their deficiency, especially if they are not exposed to sunlight. There is little evidence that adding more vitamin D to the TPN solution would have been of benefit, and it has been suggested that it could be detrimental.[12]

The fat-soluble vitamins are fairly sensitive to disturbances in lipid absorption and vitamin D malabsorption may occur even in the absence of clinical steatorrhea.[5] Vitamin D is a fat-soluble vitamin rarely found in foods naturally and is present in fortified foods such as milk and some cereals. Vitamin D can be synthesized in the skin from 7-dehydrocholesterol after exposure to UVB (wavelength, 290–315 nm) radiation.[1,2] There is strong evidence indicating that the thermal isomerization reaction from previtamin D_3 to vitamin D_3 is greatly enhanced by the lipid-membrane environment in which it is made.[13] This observation explains the rapid increase in blood levels of vitamin D_3, after exposure to simulated solar ultraviolet radiation.[10] Exposure of the body in a bathing suit to 1 MED of sunlight is equivalent to ingesting about 10,000 IU –25,000 IU of vitamin D.[3] Thus, exposure of 6%–10% of the body surface to 1 MED is equivalent to ingesting about 600–1000 IU of vitamin D.[3]

The beneficial effects of sunlight in the treatment and prevention of rickets and osteomalacia have been recognized for more than 80 years.[14] Although ultraviolet irradiation can be achieved from exposure to sunlight, it is generally available only seasonally and difficult for persons who are too ill to spend much time outdoors. Adams et al.[15] reported that exposure of normal subjects and vitamin D-deficient patients to increasing doses of UVB resulted in marked increases in the serum 25(OH)D concentrations. Kooh et al.[16] reported one infant with chronic cytomegalovirus hepatitis, and a child with atypical Alagille's syndrome who had vitamin D-deficiency rickets caused by malabsorption, and both were effectively treated with ultraviolet irradiation. In many parts of Russia where the winter is long, artificial sources such as erythemal lamps and sunlamps were widely used in large industrial and institutional buildings, and are used in public schools to prevent vitamin D deficiency in children.[17] Recently, Chunk et al.[18] exposed elderly nursing home residents to subliminal UVB radiation and showed that it prevented vitamin D deficiency. Exposure to commercially available home sunlamps with a dose schedule recommended by the manufacturers produced a significant increase in vitamin D synthesis in the skin as measured by the circulating concentrations of serum 25(OH)D.[19]

Conclusions

UVB irradiation is a very effective way to correct vitamin D deficiency in patients who have fat malabsorption. It is important to diagnose vitamin D deficiency early and treat the disease because vitamin D deficiency results in impaired bone mineralization and muscle weakness. Serum 25(OH)D and PTH levels are the only useful tests in screening for hypovitaminosis D myopathy.[9] Exposure to simulated sunlight in a tanning bed to enhance their vitamin D nutritional status may be useful for people who live in areas where the synthesis of vitamin D by natural sunlight is limited during the winter months or patients who have malabsorption of lipids. Awareness among physicians is needed for early evaluation of the vitamin D status of patients with Crohn's disease and treatment with exposure to ultraviolet irradiation when they do not respond to oral or intravenous replacement, to avoid osteomalacia or osteoporosis and its consequences.

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