Vitamin D Deficiency in an Ample Sunlight Country
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Sufficient vitamin D (25OHD) status is defined according to changes in biomarkers, which demonstrate changes in response to increase or decrease in 25OHD levels. These include parathyroid hormone (PTH), and calcium absorption. Serum 25OHD levels, lower than 20 ng/ml, is regarded as vitamin D deficiency, whereas levels between 20-30 ng/ml are called as insufficiency. These cutoffs are based on observation that intestinal calcium absorption is maximal at 32 ng/ml and PTH concentration continues to decline and reach their nadir at 30-40 ng/ml. Insufficiency is associated with elevated serum PTH levels and hence with increase in bone loss. The optimal levels for bone health are set at above 30 ng/ml.1

Recent literature reports a global pandemic of 25OHD deficiency. This deficiency affects children and healthy adults living all around the world; in the United States, Canada, Europe, Middle East, India, Australia and Asia. In Pakistanis, 25OHD deficiency was first noted in immigrants in UK in early 1970s. The expression of vitamin D deficiency after immigration to UK was assumed to be an environmental characteristic and it was thought that Pakistanis may not be getting enough sun exposure after migration.2 However, recent data shows significant problem even in sun drenched countries such as ours. Occult osteomalacia and 25OHD deficiency was reported in healthy and pregnant women and mothers and their breast-fed infants in 1976 and 1998 respectively from Pakistan.3,4 High prevalence has been described in patients including those with hip fractures, patients presenting in obstetric clinics and ambulatory care. Prevalence as high as 92% among patients has been reported from two large referral centres in Karachi. Of note is the existence of severe D deficiency in patients with tuberculosis. Poor nutritional history, lack of sunlight exposure, and low socio-economic status were identified as contributory factors. High prevalence of D deficiency in ambulatory patients has also been reported. A low serum calcium and elevated alkaline phosphatase were identified as reflective of severe deficiency, while elevated iPTH correlated with mild to moderate deficiency. Therefore, the traditional markers like serum calcium, phosphate and alkaline phosphatase were considered to be unreliable screening tools of moderate to mild deficiency. The most reliable index of vitamin D deficiency was considered to be serum 25OHD and PTH levels, which rises early in the disease process. None of the studies, so far published, has examined 25OHD status in healthy population.5-8

In two different unrelated pilot projects on healthy asymptomatic volunteers, which included both males and females from our centre, we identified 80% and 90% of the study participants to be D deficient by current criteria (unpublished data). More important was the presence of high PTH and bone turnover marker in 28% of these cases, separately in both the studies. However, the studies included ambulatory participants through convenient sampling, who consented to volunteer, adding a selection bias. The status of vitamin D deficiency was not known in the community.

The two most important causes of 25OHD deficiency are the lack of sunlight exposure and reduced intake of vitamin D containing food. The major source of 25OHD for humans is exposure to ultraviolet B (UVB) in sunlight. Any factor decreasing the penetration of UVB radiation will affect the cutaneous synthesis of vitamin D like increased pigmentation due to melanin or use of sun screen. This partly explains the high existence of 25OHD deficiency in some sunniest areas of the world. Ageing is associated with decreased concentration of 7 dehydrocholesterol, thus decreasing the availability of precursor as compared to young individuals. Being a fat soluble vitamin, it is readily taken up by the fat cells and due to this, in obesity, vitamin D is sequestered by the large body fat pool giving rise to D deficiency.1

The other important source is oral intake. However, most natural foods do not contain vitamin D in significant amount and D content is often very low to sustain healthy circulating levels of 25OHD without food fortification. It has been identified that even in countries that do fortify, vitamin D intakes are low in some groups due to their unique dietary patterns, such as low milk consumption, vegetarian diet, limited use of dietary supplements, or loss of traditional high fish intakes.1 A global review indicates that dietary supplement use may contribute 6-47% of the average vitamin D intake in some countries. From estimates of vitamin D intake, it has been found that the current food supply, supplementation practices, and dietary patterns of most countries do not adequately supply the essential...
25OHD. In case of vitamin D, for which sunlight exposure accounts for most of the vitamin D in circulation; it is important to quantify vitamin D not only from diet and supplements but also from sunlight exposure.9

The impact of deficiency on overall health of an individual is extremely large as has been demonstrated by recent researches. Vitamin D deficiency increases the risk of developing and dying from cancers of breast, colorectal, prostate and many others. In addition, the deficiency is associated with an increased risk of type I Diabetes, multiple sclerosis and hypertension. An association has also been shown with depression. In pregnancy, 25OHD deficiency has been associated with an increased risk of pre-eclampsia. Strong association has been demonstrated with tuberculosis and increased susceptibility to infections.1

The best strategy to increase vitamin D intake and to improve 25OHD status is promotion of supplementation in the group at risk and food fortification for the general population along with increasing the exposure to sunlight. Many experts now agree that in the absence of adequate sun exposure, 800-1000 IU vitamin D/day is recommended for children and adults of all ages from dietary and supplemental sources. Higher doses may be required if fat malabsorption, obesity or other causes coexist that would enhance vitamin D catabolism and its destruction.1,9

Further studies are needed to fully understand the pathophysiology of vitamin D deficiency in the South Asian population including defining the optimal levels for our population, so that appropriate preventive strategies can be introduced.10 The continuing prevalence of vitamin D deficiency in our population, particularly among adolescents, warrants immediate action. An effective preventive policy is long overdue.

REFERENCES