A. Prevention, Evaluation, and management of vitamin D deficiency in adults with developmental disabilities

Introduction and Background: Persons with severe developmental disabilities, who live in institutions or the community, are at high risk for vitamin D deficiency. This is in part due to reduced out-of-door activities, as well as associated neuro-motor dysfunction and maladaptive behaviors, such as aggression, self-injurious behavior, and PICA. When they do go outside, they are usually covered with clothing and sunscreen in order to prevent sunburn. Since most of our vitamin D is generated in the skin by UVB light conversion of 7-dehydrocholesterol to previtamin D, people with severe developmental disabilities are likely to have low bodily stores of vitamin D, especially as they age. It is been estimated that humans need 3000 to 5000 units of vitamin D per day for normal bodily function. During the winter months (November through March), 80% of our vitamin D needs are supplied by the vitamin D that has been stored during the previous summer months. Therefore 2400-4000 units per day are supplied by stores, leaving 600-1,000 units to be supplied by the diet or supplements.

Since persons with developmental disabilities are inherently making less vitamin D, most of the year-around they are vitamin D deficient. In fact, during the winter months they are at a particula great risk for vitamin D deficiency. The symptoms of severe vitamin D deficiency include muscle and bone aches and pains, weakness (including proximal myopathy that makes it difficult getting up from chairs or out of bed or to walk), increased tendency to fall, depression, and worsening COPD and seizures (due to lowered serum calcium levels). Vitamin D deficiency can also lead to Seasonal Affective Disorder. Regarding adults with a severe developmental disability who are nonverbal and cannot articulate how they feel, symptoms of vitamin D deficiency are subtle and non-specific, and vitamin D deficiency may remain undiagnosed. They may present as worsening seizures or behavior, clumsiness, falls and injuries, or "cabin fever “.

The second major cause for vitamin D deficiency in this highly vulnerable population is administration of drugs that interfere with vitamin D metabolism. This could be secondary to psychotropic or anti-convulsant drugs which affect the conversion of vitamin D to 25 hydroxy vitamin D [25(OH)D] in the liver. In the presence of renal insufficiency, production of the active metabolite, 1,25 dihydroxy vitamin D [1,25(OH)2D] is also impaired. In the latter scenario, 25(OH)D levels that are measured to assess vitamin D status, may be normal, but the 1,25(OH)2D are low leading to significant secondary hyperparathyroidism.

Suggested strategy for the treatment and prevention of hypovitaminosis D in adults with developmental disabilities:

There are numerous strategies for the treatment and prevention of hypovitaminosis D. Perhaps the most well-known approach also endorsed by the American Endocrine Society, is to first administer a loading dose of 50,000 international units per week for 8 to 12 weeks followed by a maintenance dose of 50,000 units per month, perhaps, indefinitely. This approach has been shown to be both safe and effective in
treating vitamin D deficiency and preventing its recurrence. The optimal serum level of vitamin D is under debate, but many endocrinologists feel that 30 to 50 ng/mL is safe and efficacious in preventing diseases.

When evaluating a person with a severe developmental disability for vitamin D deficiency for the first time, many patients may already be receiving 400 to 600 international units of vitamin D through their diets. More than half of persons with a severe developmental disability taking psychotropic and/or seizure medication may have a low or low-normal serum level of 25(OH)D. Most of these patients also have low bone mineral density and increased bone turnover as determined by elevation of biochemical markers of bone turnover, and very high incidence of fractures.

In this group of patients, it makes sense to give therapeutic doses of vitamin D until serum vitamin D level increase above 30 ng/mL, and then put them of a reasonable maintenance dose. If necessary, the loading dose can be repeated, especially to replenish tissue vitamin D stores. Candidates for this approach would include individuals with hypovitaminosis D levels below the normal range of 30 ng/ml, as well as those who are have low BMD, are receiving seizure or anti-psychotic medications, and/or are at risk for falls.

Studies have shown that older people who are at risk for falls and patients receiving seizure medication, can benefit from long-term adequate vitamin D supplementation. These patients respond with increased bone mineral density and reduction of falls and fractures, even when pretreatment vitamin D blood levels are low normal.

A maintenance dose could be 50,000 units per month initially. Monitoring may be accomplished by measuring serum 25(OH)D levels at six months after stabilization, and then once yearly. In the case of patients with severe developmental disability (MCDD), it seems appropriate to keep the serum vitamin D levels between 40 to 50 ng/mL, while keeping serum calcium level in the normal range.

Follow-up markers of bone turnover such as P1NP and CTX should also be determined in order to determine the state of bone turnover. If bone mineral density is low and bone turnover continues to be elevated despite a maintenance dose of vitamin D, a pharmaceutical agent should be considered, since aging adults with severe developmental disabilities are at very high risk for low bone mineral density and fracture. However, it is critical to correct the underlying vitamin D deficiency prior to that.

Correction of vitamin D deficiency is an important strategy in the treatment of states of low bone mineral density, falls, and neuro-motor dysfunction. It is important that the treating physician take a comprehensive approach to ensure the optimal treatment of physiological vitamin D deficiency in adults with severe developmental disabilities.

References
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Low bone mineral density (BMD) is commonly determined by Dual-Energy X-ray Absorptiometry (DXA). Clinical experience and published reports have shown that low BMD is especially common in adults with developmental disabilities, such as mental retardation, epilepsy, autism, and cerebral palsy. Regardless of the cause of the developmental disability, low bone mineral density occurs in as many as 80%, or more in some studies. Having low BMD increases the risk for fracture and, not surprisingly, the incidence of fracture in adults with developmental disabilities is increased as well.

The causes of low BMD in adults with developmental disabilities include 1) failure to obtain a normal peak bone mineral density during young adulthood, 2) lack of adequate regular weight-bearing exercise, 3) immobility secondary to neuromotor dysfunction, 4) the adverse effects of various drugs (e.g. neuroleptics, SSRIs, lithium, antiepileptic medication, and proton pump inhibitors), and 5) vitamin D deficiency.

Strategies to increase BMD in adults with developmental disabilities must include: 1) measures to promote weight-bearing exercise, 2) elimination of unnecessary drugs, 3) provision of optimal amounts of vitamin D and calcium, and 4) the use of bone-active pharmaceutical agents (as a last resort).

Special attention should be paid to the usage of psychotropic and seizure medications. Efficacy of these classes of drugs should be carefully documented and if they are demonstrated to not be absolutely necessary, they should be tapered and discontinued. Drug-Related Bone Loss (DRBL) is probably a major cause of low BMD encountered in adults with developmental disabilities, and efforts to eliminate polypharmacy may provide enormous benefits with regards to reduction in fracture risk in this special population of adults.

Optimization of vitamin D and calcium is another important strategy that has the potential to increase bone mineral density in adults with developmental disabilities. When maximally stimulated by the sun the skin has the capacity to produce 10,000 to 20,000 international units of vitamin D per day. Adults need approximately 3000 to 5000 units per day to support normal metabolic functions (almost all cells have vitamin D receptors). During the winter months (October through April), 80% of the daily needs for vitamin D come from vitamin D stores (2400 to 4000 IU's/day) that were stored during the summer months (May through September). Thus 20% (600 to 1000 international units/day) is needed to be supplied by the diet or supplements in order to avoid vitamin D deficiency during the winter months.

However, many people with developmental disabilities, especially those who reside in institutions, have little outdoor activity during the summer months and when they do have outdoor activities tend to be covered with sunblock and clothing in order to prevent sunburn. Therefore adults with developmental disabilities most likely have very low stores of vitamin D and are at great risk for vitamin D deficiency especially during the winter months. Of course those who are in addition receiving drugs which interfere with vitamin D metabolism are at an even greater risk for vitamin D deficiency.
In the process of optimizing vitamin D for adults with developmental disabilities, it probably makes sense to begin with a loading dose of vitamin D in order to replenish vitamin D stores. A common regimen that has been shown to be safe and effective is to begin with 50,000 international units of vitamin D3/ per week for 8 to 12 weeks, and then continue a maintenance dose of 50,000 units per month (1-3). There does not appear to be a universal agreement on the best target blood level but a 25 hydroxy vitamin D level of 40 to 50 ng per ML is probably reasonable (4).

It should also be determined that hypercalcemia does not occur, and that bone turnover markers are not elevated. Vitamin D and calcium alone may be sufficient to correct low bone mineral density encountered in some adults with developmental disabilities, but others may require a pharmaceutical agent. If a pharmaceutical agent is required, it remains critical that vitamin D be optimized before the drug is started.

References


