Diminished Stress Resilience in Institutionalized Elderly Patients: Is Hypovitaminosis D a Factor?

SIR: Epidemiological research suggests that hypovitaminosis D has reemerged as a problem in climatically diverse regions worldwide, even in mid-latitude, temperate regions such as California and Australia.[1,2] Thomas et al.[1] have provided strong evidence for an increased risk of hypovitaminosis D even among inpatients whose dietary intake of vitamin D meets or exceeds national standards.

Although hypovitaminosis D contributes to the still surprisingly high incidence of rickets in the United States,[2] its greatest impact is among elderly persons and medical inpatients.[1] Initial symptoms of hypovitaminosis D are diminished resilience to psychosocial stress, generalized muscle pain, and nonspecific fatigue.[1,2] The classic symptom of hypovitaminosis D, osteomalacia (and the associated increased risk of hip fracture), usually only becomes evident in later stages.[1,2]

Ultraviolet (UV) light is an essential factor in the production of the active form of Vitamin D (Vitamin D hormone; VDH).[1] Therefore, low sun exposure is a major risk factor for hypovitaminosis D.[1,2] Other risk factors include advanced age, chronic liver and renal diseases, and a low-fat diet.[1] The increased risk of hypovitaminosis D in elderly and chronically mentally ill persons may be largely a result of their overrepresentation in housebound and institutionalized populations.[1]

Current medical recommendations, such as avoiding sunlight to lower the risk of skin disease and decreasing fat intake to lower the risk of coronary heart disease, may also be contributing to the reemergence of hypovitaminosis D.[1,2] Although these standard recommendations should not be reversed, such preventive health measures may be contributing to an increased incidence of hypovitaminosis D.

Accumulating evidence in animal models suggests that hypovitaminosis D increases the risk for glutamate excitotoxicity during acute activation of the fear circuitry and that systemic vitamin D attenuates oxidative injury to the locus coeruleus.[3] These results suggest that hypovitaminosis D may be linked to lower resilience to acute psychosocial stress in humans. This vulnerability may also extend to the effects of chronically elevated allostatic load.

Drugs such as phenytoin, carbamazepine, and rifampin have been shown to interfere with vitamin D activation or clearance, and research is examining whether newer mood stabilizers, such as valproate and lamotrigine, interfere with vitamin D activation.[2,4] Supplementation should be considered in any high-risk group. Laboratory assays for VDH are widely available but rarely utilized and can easily be included in psychiatric research protocols alongside glucocorticoid and mineralocorticoid levels.

In summary, hypovitaminosis D may interact negatively with the stress response, and institutionalized and housebound elderly patients are at higher risk for low VDH levels. If confirmed, this idea is especially noteworthy because, unlike many other risk factors affecting stress-related morbidity, treatment of hypovitaminosis D is simple, inexpensive, and likely to have high patient acceptance.

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