Obesity, Influenza Virus Infection, and Hypovitaminosis D

O’Brien et al [1] found that obesity was a risk factor for severe influenza infection in mice and proposed poor lung wound healing, leading to pulmonary edema suggestive of acute respiratory distress syndrome, as a potential underlying mechanism, rather than a lack of viral control. They suggested that obesity should be recognized as a risk factor requiring early antiviral therapy [1]. However, the potential effect of hypovitaminosis D in this context was not considered, nor the consequent therapeutic role of vitamin D in normalization.

The classical role of vitamin D is to regulate calcium homeostasis. However, it has become clear that the extraskeletal health benefits of vitamin D are numerous. In particular, 1,25-dihydroxyvitamin D, the biologically active form of vitamin D, has been shown to be a potent immune modulator of the adaptive immune system and to stimulate the innate immune response after infection [2]. The proper functioning of the body’s defense system requires the presence of adequate levels of vitamin D for barrier integrity, the production of antimicrobials, chemotaxis of other immune cells, and regulation of inflammation in the innate and adaptive immune system [3]. These findings may be particularly important for the prevention of influenza and its related complications.

Indeed, it is well known that seasonal influenza strikes in the northern hemisphere during the winter when vitamin D levels are lowest as a result of less UVB radiation. A prospective study [4] correlated serum 25-hydroxyvitamin D [25(OH)D] concentrations with the incidence of viral respiratory infection among adult women and men. During 114 days of the fall and winter in a temperate zone, a serum concentration of 25(OH)D of ≥38 ng/mL was associated with a 2-fold decrease (P < .0001) in the risk of developing acute viral infection of the respiratory tract [4]. Furthermore, a recent randomized control study involving school children receiving 1200 IU of vitamin D for the winter months resulted in a significant reduction (relative risk, 0.58) in developing influenza A during the influenza season [5].

In contrast, mounting evidence suggests that adiposity is inversely associated with serum 25(OH)D levels [6, 7]. It has been proposed that the association between hypovitaminosis D and obesity may be related to decreased exposure to sunlight because of limited mobility and vitamin D sequestration in body fat compartment [8]. Moreover, it has been found that adiposity measures, including body mass index, waist-to-hip ratio, body fat and trunk fat, predicted increased incident vitamin D deficiency, and decreased likelihood of recovery from vitamin D deficiency [9]. Of note, severely obese individuals with and without chronic conditions have been found to be at increased risk for respiratory hospitalizations during influenza seasons [10].

Thus, it might be plausible that the results by O’Brien et al are attributable to low serum 25(OH)D concentrations in obese mice. It would be easy to do an experiment to test this hypothesis by comparing results for vitamin D-deficient mice with vitamin D-replete mice. These data could provide direction for future interventional studies in humans examining the efficacy of vitamin D supplementation in reducing the incidence and severity of influenza, in the general population and in specific subpopulations at risk for hypovitaminosis D, such as obese persons.

Note

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