Vitamin D Deficiency Is More Common in Type 2 Than in Type 1 Diabetes

We report data from a pilot study examining vitamin D deficiency in type 1 and type 2 diabetes. Serum 25-OH vitamin D (25-OH-D) levels were measured in type 1 (n = 50) and type 2 diabetic (n = 63) individuals at the time of their routine visits for diabetes management. Patients had no prior history of metabolic bone disease, vitamin D deficiency, parathyroid disease, malabsorption, or significant elevations of serum creatinine or liver enzymes. Most subjects (74.3%) were prescribed a daily multivitamin as part of their routine diabetes care, but none were taking additional supplements. Serum was obtained during “light” (April to September) and “dark” (October to March) months. Serum 25-OH-D levels were measured using a competitive protein-binding assay (Esoterix, Calabasas Hills, CA). Vitamin D levels were classified as deficient (0–20 ng/ml) or expected (>20 ng/ml). Although 25-OH-D levels >32 ng/ml are optimal (1), levels ≥20 ng/ml are usually considered acceptable and are generally not treated in patients without metabolic bone disease.

Type 1 diabetic individuals were significantly younger than those with type 2 diabetes (mean ± SE 49.0 ± 1.7 vs. 61.2 ± 1.5 years, P < 0.001) and had a lower BMI (26.2 ± 0.7 vs. 33.9 ± 1.0 kg/m², P < 0.001). The majority (63.5%) of the type 2 diabetic individuals were deficient compared with 36% of the type 1 diabetic patients. The unadjusted mean 25-OH-D level for type 2 diabetic patients was 17.1 ± 1.2 ng/ml (P < 0.05) compared with 23.6 ± 1.2 ng/ml for type 1 diabetes (P < 0.05). Of the patients who were deficient, most were prescribed multivitamins (72.2% of type 2 diabetes, 72.5% of type 1 diabetes). Most patients having serum 25-OH-D levels >20 ng/ml were also prescribed multivitamins (73.9% of type 2 diabetes, 78.1% of type 1 diabetes). Serum was obtained with equivalent distribution in light and dark months among both groups.

The 25-OH-D levels inversely correlated with BMI (P < 0.01) but were not directly related to age or sex. ANCOVA was used to compare 25-OH-D levels in type 1 and type 2 diabetic patients while adjusting for BMI and age. As was noted in the unadjusted levels, significantly lower 25-OH-D levels were found in type 2 relative to type 1 diabetic patients (mean adjusted 25-OH-D 18.1 ± 1.4 vs. 22.9 ± 1.6 ng/ml, P < 0.001). Among type 2 diabetic patients, mean 25-OH-D level was similar in those taking insulin compared with those on oral hypoglycemic agents (mean 25-OH-D 17.6 ± 1.6 and 16.8 ± 1.5 ng/ml, respectively; P = 0.73).

Our findings suggest that vitamin D deficiency is more common in type 2 diabetes than in type 1 diabetes, unrelated to age, sex, or insulin treatment. Although higher BMI was associated with lower 25-OH-D levels, the difference in vitamin D levels between type 2 and type 1 diabetic individuals persisted after adjusting for BMI. This difference may be therapeutically significant in that mean vitamin D levels in type 2 diabetic patients were in the deficient range whereas mean levels in the type 1 diabetic patients reached the expected level. Despite most type 2 diabetic patients being prescribed a daily multivitamin, usually containing 400 IU of vitamin D, the percent who were deficient remained unchanged. Further studies are needed to better understand the causes and clinical significance of the observed hypovitaminosis D and to investigate response to vitamin D replacement therapy.

Circulating Levels of Interleukin-18 Independent of Body Fat and Fat-Free Mass

Results from the MONICA/KORA study

Elevated systemic concentrations of interleukin (IL)-18, a potent proinflammatory cytokine, have recently been reported to increase the risk of developing type 2 diabetes in a prospective case-cohort study with a mean follow up of 10.8 years (1). IL-18 is secreted from adipocytes (2), and IL-18 levels were found to be increased in obesity and decreased after weight loss in obese women (3). However, we did not observe a direct relationship between BMI and IL-18 levels in a large population-based sample (1). Another recent study in 144 men surprisingly reported a significant association of IL-18 with fat-free mass (4). We now present a detailed analysis of the relationship of IL-18 serum concentrations with body composition and potential confounding effects by sex in 225 men and 230 women from the MONICA (Monitoring of Trends and Determinants in Cardiovascular Disease)/KORA (Cooperative Health Research in the Region of Augsburg) Survey 1994/1995 (1), for whom bioelectric impedance data were available.

Serum IL-18 concentrations did not differ between men and women (geometric means 154.4 and 163.9 pg/ml, respectively; P = 0.38). For men and women combined, log IL-18 was not significantly correlated (Pearson) with absolute fat mass (r = -0.02; P = 0.58), percent fat mass (r = 0.01; P = 0.82), and fat-free mass (r = -0.04; P = 0.38). Separate analyses for men and women yielded similar results. The strongest correlations were seen between log IL-18 and systolic blood pressure (r = 0.11; P = 0.01), diastolic blood pressure (r = 0.11; P = 0.01), C-reactive protein (r = 0.09; P = 0.054), and log IL-6 (r = 0.10; P = 0.02), whereas correlations with BMI, waist circumference, cholesterol levels, and uric acid did not reach statistical significance.

Taken together, these data show that systemic IL-18 concentrations are independent from body fat composition in-
The Metabolic Syndrome: Time for a Critical Appraisal: Joint Statement From the American Diabetes Association and the European Association for the Study of Diabetes

We read with interest the American Diabetes Association statement about the metabolic syndrome (1). The idea that the aggregation of borderline risk factors could result in cardiovascular damage equal or superior to that occurring in individuals carrying a full-weight risk factor disease was intriguing. However, this intuition was soon polluted by inclusion of patients with frank diseases. If the philosophy of the metabolic syndrome is to draw firm attention upon the way to disseminate healthy practices and to trace the natural history of the syndrome, then the time is to avoid double-labeled diagnosis, for example diabetes and the metabolic syndrome; to give the real number of subjects at risk and to interfere with its evolution with lifestyle or pharmacological interventions. In the National Health and Nutrition Examination Surveys III sample, ~8% of coronary heart disease events occurred in people with only borderline levels of multiple risk (2). Moreover, intensive lifestyle intervention (3), diet (4), and drugs (5) have all been shown to be effective in reducing the prevalence of metabolic syndrome, although interventions based on diet, physical activity, and weight reduction seem to work better than drugs. Lastly, it would be easier to force a labeled patient (that with a pure syndrome) to follow advice for lifestyle changes than for unlabeled subjects with one or more borderline risk factors. Since the way to disseminate healthy practices is all but easy, any help is welcome.

References