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Vitamin D and type 1 diabetes, the big mistake and the hope for primary and secondary prevention

Dimitrios T. Papadimitriou
 Attikon University Hospital, Greece

The role of Vitamin-D in innate and adaptive immunity is critical. Redirection of human autoreactive T-cells upon interaction with dendritic cells can be modulated by an analog of 1,25-dihydroxyvitamin D₃. Moreover, T1D autoantibodies can be “negativated” with oral calcitriol. Based on recent knowledge of the possible involvement of 1,25-dihydroxyvitamin D in the pathogenesis of type 1 diabetes (T1D) and the results of its administration in animal models, we conducted a clinical trial by treating high-risk children, positive for T1D autoantibodies, with oral calcitriol. Daily calcitriol 0.25 mg effectively negativates anti-GAD65 antibodies and IAA after a median time of 6 months. This simple, safe, and low-cost strategy may prove effective in the prevention of T1D in the future (1). Expanding our efforts for secondary prevention using High

Doses of Oral Calcitriol (up to 6µg/day) and Paricalcitol (up to 72 µg/day) we have reported a successful interception to the progression to clinical disease for over 3 years in a 10 yr-old boy with Type 1 Diabetes (2). Since 2006, T1D in Finland has been decreasing after an initial plateau preceded by an increase in serum25OHD after the authorities’ decision for fortification of dietary milk products with cholecalciferol. A statistical error in the estimation of the Recommended Dietary Allowance (RDA) for VitaminD was recently discovered, indicating that 8895 IU/day are needed for 97.5% of individuals to achieve values ≥ 50 nmol/l, analyzing correctly the same data used by the Institute of Medicine (3).

info@pedoendo.gr

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