Endocrinología, Diabetes y Nutrición

226 - MENOPAUSE: AN ADDITIONAL RISK FACTOR FOR TYPE 2 DIABETES IN PREDIABETIC WOMEN?

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Resumen

Introduction: Menopause may be an additional risk factor for type-2 diabetes (T2DM) in prediabetic women, although its relationship with prediabetic markers has not been complete elicited.

Methods: This study followed a cohort of 229 prediabetic females for 5.5 years, comparing T2DM occurrence in postmenopausal women (#1 51 years-old, according to the Spanish Society of Gynecology and Obstetrics-SEGO) with premenopausal women, and was associated with prediabetic markers and clinical parameters.

Results: Baseline data revealed that prediabetic women (average age 52.7 yrs, BMI = 30.3 ± 6.73) exhibited impaired fasting glucose (IFG, 48%), impaired glucose tolerance (IGT, 54.6%), and elevated HbA_{1c} (70.7%). Hypertension (42.8%), hypothyroidism (28.4%), and low vitamin D levels (17.7 ng/mL) were also observed. Postmenopausal women showed significantly higher HbA_{1c}, hypertension, liver enzymes, fibrosis-4 index, uric acid levels, and lower glomerular filtration rate compared to premenopausal women. Interestingly, 26% of postmenopausal-age women developed T2DM in 2.3 yrs, while 19.7% of premenopausal-age did it. Among postmenopausal women with T2DM, higher BMI, IFG, IGT, hypertension, uric acid, and lower HDL-C and renal function were exhibited compared to non-diabetics, while premenopausal diabetics only showed higher BMI. Cumulative T2DM incidence in postmenopausal women remained similar according to prediabetes criteria (30% with IFG, 29.6% with IGT, and 25.9% with HbA_{1c} #1 5.7%). Crude analysis associated postmenopausal women with T2DM debut to IFG and IGT, but only IGT remained significant after adjustments for age and BMI (RR = 2.29, p = 0.043). In premenopausal women none of the prediabetic marker associated with T2DM.

Conclusions: Menopause may proposed as a risk-factor for T2DM in prediabetic women, particularly influencing IGT-associated incidence. Contributing factors such as uric acid and renal function appear to create distinct prediabetic phenotypes for T2DM development in those.