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Prognostic value of body mass index and waist circumference in patients with chronic heart failure (Spanish REDINSCOR Registry)

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Propose: To analyze the prognostic value of Body Mass Index (BMI) and Waist Circumference (WC) on total mortality and cardiac death in patients with chronic heart failure in a national multicenter registry.

Methods: The study included 2,254 patients with heart failure who were followed-up for 4 years (mean age 66.7 ± 10.1 years). Obesity was classified as BMI ≥ 30 kg/m2 and overweight as WC ≤ 88 cm in women or ≥ 102 cm in men. Independent predictors of total and cardiac mortality were assessed using a multivariate Cox model. The final model was adjusted for variables (age, sex, comorbidities) as clinically relevant and confounding variables were included when they carry a change on the hazard ratio greater of 5.

Results: Obesity was present in 35% of patients, overweight in 43%, and central obesity in 60%. Patients with obesity and central obesity both had a higher percentage of women, incidence of hypertensive or hypertrophic etiologies, diabetes, dyslipidemia and hypertension (p < 0.05). In contrast, patients with normal weight were more anemic, had worse NYHA functional class, more depressed TSH levels and higher levels of NT-pro-BNP (p < 0.05). During follow-up there were 475 deaths (72%) were cardiac). Obese and overweight patients had lower total mortality (16.4% and 21.3%) than patients with normal weight (28.4%, p < 0.0001). Similarly, patients with higher WC had a lower total mortality than patients with normal WC (18.9% vs. 24.3%, p = 0.002). Cardiac mortality was lower in obese and overweight patients (12.3% and 15.4%) than in patients with normal weight (19.3%, p = 0.004). Likewise, patients with higher WC presented a lower cardiac mortality than patients with normal WC (13.9% vs. 17.1%, p = 0.038). In a Cox model adjusted by sex, age, etiology, diabetes, anemia, previous AMI and confounding factors, BMI and WC were independent predictors of lower total mortality. Hazard Ratio (HR) > 0.84, 95% confidence interval (CI): 0.76-0.92, p < 0.001, and WC > 97 cm, 95% CI: 0.94-0.99, p = 0.012 respectively). Also were predictors of lower cardiac death (BMI HR=0.84, 95% CI: 0.75-0.93, p < 0.001 and WC HR=0.97, 95% CI: 0.94-0.99, p=0.012). An interaction effect between BMI and WC was found (HR=1.01, 95% CI: 1.001-1.002, p =0.002) and the protective effect of BMI was lost when patients had a WC greater than 120 cm.

Conclusions: This study confirms the paradox of a reduction of total and cardiac mortality in patients with increased BMI and WC. Moreover, data show that this protection is lost when the BMI or the WC attain extreme levels.

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Thirty year risk of cardiovascular disease and key risk factors increase with increasing thyroid stimulating hormone in adolescents

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Purpose: To investigate the dose-response association between Thyroid Stimulating Hormone (TSH) level in serum (μU/ml) and 30-year risk of Cardiovascular Disease (CVD) and its key risk factors in adolescents.

Methods: A cohort of 562 participants from the Sopkard 15 regional prevention program (mean age 14.1±0.1 years) had their TSH level, blood pressure and lipid profile measured. Risk factor data was used to estimate the 30-year risk of CVD according to the Framingham algorithm as an aggregate measure of risk factor burden. The relationship between TSH and the 30-year risk of CVD as well as its key risk factors (systolic blood pressure, total and HDL cholesterol, triglycerides and body mass index) was modeled using penalized regression splines with three knots located at the 5th, 50th and 95th percentiles of the distribution of TSH and adjusting for age, sex and smoking.

Results: We observed statistically significant monotone increasing relationships between TSH level and 30-year risk of CVD (p-value = 0.024), systolic blood pressure (p-value = 0.010), total cholesterol (p-value = 0.012) and triglycerides (p-value = 0.001). The relationship did not reach statistical significance for body mass index and HDL cholesterol. 30-year risk increased more rapidly with TSH levels in the range 0 – 3 μU/ml and leveled off for TSH levels above 3 μU/ml.

Conclusions: Aggregate and individual burden of CVD risk factors increases with increasing levels of TSH in adolescents. The monotone nature of the relationship suggests that from the perspective of CVD risk burden any threshold used to define the normal range of TSH remains arbitrary.