
Sex hormone binding globulin level is inversely associated with presence and severity of abdominal aortic calcification in women but not men in the Multi-Ethnic Study of Atherosclerosis (MESA)

ED Michos, D Vaidya, Johns Hopkins University, Baltimore, MD; SM Gapstur, Northwestern University, Chicago, IL; PJ Schreiner, University of Minnesota, Minneapolis, MN; SH Golden, Johns Hopkins University, Baltimore, MD; ND Wong, University of California at Irvine, Irvine, CA; MH Criqui, University of California, San Diego, San Diego, CA; P Ouyang, Johns Hopkins University, Baltimore, MD

Background: Endogenous sex hormones (SH) may influence the development of atherosclerosis. Conflicting findings exist regarding the associations of total testosterone (T), estradiol (E2), and dehydroepiandrosterone (DHEA) with subclinical atherosclerosis, while most studies show a protective effect of SH binding globulin (SHBG) in both postmenopausal women and men. SH are also associated with lipoprotein levels. Because cardiovascular disease (CVD) risk differs by sex, we examined if the relation of SH with abdominal aortic calcium (AAC) differs between men and women.

Methods: We analyzed cross-sectional data of 881 postmenopausal women and 978 men from the baseline visit of MESA, a multicenter NHLBI study of participants free of clinical CVD, who had both AAC quantified by computed tomography and SH levels assessed (T, E2, DHEA, and SHBG). We examined the association of log (SH levels) with presence of AAC by logistic regression, and in subjects with AAC, determined the association with log (AAC) extent using linear regression. For each SH, we adjusted for covariates of age, race, hypertension, smoking, diabetes, BMI, hormone replacement therapy [women only], and the other SH (Model 1) and then additionally adjusted for total cholesterol /HDL ratio and use of cholesterol medications (Model 2).

Results: AAC was present in 73% of men (mean age 62 + 10 years) and 74% of women (mean age 64 + 9 years). For women, Model 1 showed an inverse association of SHBG with both AAC presence (OR 0.61, 95% CI 0.42 to 0.90) and extent (0.30% lower AAC score for every 1% higher SHBG level, $\beta = -0.30$ [95% CI -0.58 to -0.015]). After further adjustment for cholesterol (Model 2), SHBG was no longer independently associated with either presence or extent of AAC. In men, we found no association of SHBG with either presence or severity of AAC in Models 1 or 2. There was no independent association of T, E2, or DHEA with presence or extent of AAC in either men or women in Models 1 or 2.

Conclusion: SHBG levels are inversely associated with both the presence and severity of AAC in women but not in men after adjustment for non-lipid CVD risk factors. The association of SHBG with AAC in women may be accounted for by lipoprotein levels.