Metabolic Aspects and Mechanisms

9.26 Diagnosis of Metabolic Syndrome Based on History of Exposure or Present Abnormalities: Evidence of Necessity of a Better Definition

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Introduction. Metabolic syndrome (MetS) definition is based on either abnormal values, or ongoing therapy demonstrating history of exposure to risk factors. This classification implies that therapy does not modify the MetS status, whereas therapy-modified components might modify attribution to, and prognosis of MetS. We tested the hypothesis that definition of MetS based on history rather than on direct evidence of abnormal risk factors is associated with a more benign cardiovascular (CV) phenotype.

Methods. We studied 1642 African-American (1045 women) and 1289 Caucasian (681 women) HyperGEN participants, without prevalent CV disease or renal failure (serum creatinine >2.5mg/dL). MetS was defined by ATPIII, using cut-offs of blood tests, waist girth and measured blood pressure (BP), irrespective of on-going therapy (M1). M1 was compared with a subgroup of participants classified as MetS, based on history of exposure and on-going therapy to normalize risk factors (M2). We used LV mass index (LVMI), relative wall thickness (RWT) and a the ratio of stroke volume to pulse pressure (SV/PP) adjusted by heart rate, body weight and age (prognostically validated as a raw estimate of arterial compliance), as measures of end-organ damage.

Results. Among 1102 participants with MetS (38%), 780 were confirmed by M1, whereas 322 (15%) had MetS by M2; 1892 were free of MetS by both criteria (no-MetS). Among M2 participants, 67% had normal fasting glucose, 71% normal triglycerides, 32% normal HDL-cholesterol, 16% normal waist girth and only 4% had normal BP. After adjusting for age, sex and ethnicity, mean LVMI was higher with in M1 (41.9 g/m^{2.7}) than in M2 (39.9 g/m^{2.7}) or no-Mets participants (37.6g/m^{2.7}; all p<0.01). Similarly, RWT was higher in M1 (0.35) than in both M2 and no-MetS (both 0.34; both p<0.003). Also SV/PP was lower in MetS by M1 than in either no-MetS or MetS by M2 (both p<0.0001).

Conclusions. Diagnosis of MetS based on real-time abnormal risk factors is associated with more adverse CV phenotype than found in subjects with history of exposure corrected by treatment. The correction of MetS in some subjects is due to normalization of metabolic abnormalities, possibly due to concomitant therapy, with BP frequently suboptimal, and is associated with a more benign CV phenotype, intermediate between MetS and no-MetS.