

J Hum Hypertens. 2007 Aug;21(8):625-32. Epub 2007 May 3.

Clusters of metabolic risk factors predict cardiovascular events in hypertension with target-organ damage: the LIFE study.

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Abstract

The relation of metabolic syndrome (MetS) with cardiovascular outcome may be less evident when preclinical cardiovascular disease is present. We explored, in a post hoc analysis, whether MetS predicts cardiovascular events in hypertensive patients with electrocardiographic left ventricular hypertrophy (ECG-LVH) in the Losartan Intervention For Endpoint (LIFE) reduction in hypertension study. MetS was defined by ≥ 2 risk factors plus hypertension: body mass index ≥ 30 kg/m², high-density lipoprotein (HDL)-cholesterol $< 1.0/1.3$ mmol/l ($< 40/50$ mg/dl) (men/women), glucose ≥ 6.1 mmol/l (≥ 110 mg/dl) fasting or ≥ 7.8 mmol/l (≥ 140 mg/dl) nonfasting or diabetes. Cardiovascular death and the primary composite end point (CEP) of cardiovascular death, stroke and myocardial infarction were examined. In MetS (1,591 (19.3%) of 8,243 eligible patients), low HDL-cholesterol (72%), obesity (77%) and impaired glucose (73%) were similarly prevalent, with higher blood pressure, serum creatinine and Cornell product, but lower Sokolow-Lyon voltage (all $P < 0.001$). After adjusting for baseline covariates, hazard ratios for CEPs and cardiovascular death (4.8+/-1.1 years follow-up) were 1.47 (95% confidence interval (CI), 1.27-1.71)- and 1.73 (95% CI, 1.38-2.17)-fold higher with MetS (both $P < 0.0001$), and were only marginally reduced when further adjusted for diabetes, obesity, low HDL-cholesterol, non-HDL-cholesterol, pulse pressure and in-treatment systolic blood pressure and heart rate. Thus, MetS is associated with increased cardiovascular events in hypertensive patients with ECG-LVH, independently of single cardiovascular risk factors.

PMID: 17476291 [PubMed - indexed for MEDLINE]