A Dangerous Cocktail for Older Women?

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Cardiovascular disease (CVD) remains the leading cause of death for men and women despite substantial progress in medical and surgical treatment. Because the life expectancy in women is greater than that of men, the resulting burden of CVD in older women is a major public health issue. With the rising rate of obesity, it is anticipated that the incidence and prevalence of hypertension will continue to increase in both men and women during the next decades. Despite the proven efficacy of antihypertensive drugs, dietary and behavioral modification, and the overwhelming evidence supporting the increased risk of CVD in hypertensive subjects, why is it that hypertension remains a major public health issue worldwide?

The lack of an adequate answer to this key question is a consequence of the complex nature of the hypertensive phenotype and the interplay across various factors that influence the management of hypertension. One potential explanation is that comorbid conditions may exert an important role through their synergistic effects on CVD risk or their influence on the management of hypertension. Among co-morbid conditions, type 2 diabetes is associated with a 2- to 3-fold increased risk of CVD. The underlying insulin resistance is known to cluster with other metabolic derangements, including dyslipidemia and higher levels of inflammatory cytokines. In 1988, Raevens described the concept of the metabolic syndrome (MS) as a cluster of hypertension, insulin resistance or glucose intolerance, abdominal obesity, and atherogenic dyslipidemia resulting in a prothrombotic and proinflammatory state. MS is highly prevalent in the United States (25% to 35% depending on the chosen definition) and is associated with a 2- to 4-fold increased risk of CVD and mortality and a 5-fold increased risk of type 2 diabetes. Could the coexistence of hypertension and MS be a deadlier cocktail for CVD risk than either condition alone in older women?

In the current issue of Hypertension, Rossi et al examined intermediate factors and not CVD hard end points such as stroke, myocardial infarction, and heart failure, one could presume that the coexistence of MS and hypertension would lead to a more than an additive effect on those end points. An important message is that clinicians should not only monitor body weight but also encourage their patients to maintain a healthy weight, especially in the presence of hypertension. In addition, an aggressive management of hypertension and overweight/obesity in the presence of MS would appear to be appropriate in such patients. Finally, it is essential to curb the obesity epidemic through multifactorial interventions, including a healthier diet, physical activity, and surgical intervention where appropriate, among other measures.
Despite those clinical and public health implications arising from the current study, it remains uncertain whether the coexistence of MS and hypertension among postmenopausal women affects the bottom line. The lack of comparative data on mortality and major hard end points limits the utility of these findings. Because MS subjects used more antihypertensive agents than subjects without MS, it is possible that poor compliance, as is expected with an increasing number of drugs among MS subjects, could partially account for the poor treatment effect in the MS group. Furthermore, a differential compliance in lifestyle factor recommendations and intergroup differences in genetic predisposition, severity of hypertension, and other factors between the 2 groups may have played a role in these findings. In light of these limitations, it remains important to examine in future studies whether there is a synergistic effect between MS and hypertension on the risk of CVD and mortality and to quantify the magnitude of such effect in postmenopausal women. Such confirmation would call for a focus on global CVD risk appraisal and an aggressive management of underlying comorbidity.

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References
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