C-Reactive Protein and the Risk of Developing Hypertension

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INTRODUCTION

Hypertension has long been recognized as a significant mortality risk factor. Certain risk factors have been established to be predictors of hypertension including family history and obesity. However, no single marker has been isolated as a foolproof sine qua non test that is predictive of later development of hypertension. In this study, researchers attempt to see if there is positive correlation between C-reactive protein and subsequent development of hypertension.

METHODS

The Women’s Health Study is a double-blind ongoing study of women, age 45 and above, to determine if low dose aspirin and Vitamin E can prevent cardiovascular disease and cancer. The study began in 1992 and is still in progress. As part of the protocol, base-line serum samples were obtained at time zero and frozen.

In this study, frozen serum from 1992 was tested for the presence of C-reactive protein. A total of 20,525 women were part of the study in that they were found to be without hypertension at time zero. These women were then examined for the subsequent development of or current presence of hypertension. Results were corrected for obesity, smoking, exercise, alcohol use, parental history of MI before age 60, diabetes, hyperlipidemia, and postmenopausal hormone replacement treatment.

RESULTS

A total of 5,365 cases of hypertension were discovered during a follow-up of 7.8 years. This represents a 26% rate of hypertension development. There was a positive association between increasing C-reactive proteins
and risk of developing hypertension as shown in the Table.

**DISCUSSION**

The results of this study would suggest that there is a positive correlation from an elevated C-reactive protein and the eventual development of hypertension in women over the age of 45. In addition, there appears to be a linear relationship with the higher CRP levels, the greater the chance of hypertension to develop later in life. In those women who had a modest elevation of CRP (>1 mg/L, but <3 mg/L), the relative risk was approximately 16% higher. However, in those women whose CRP levels were 3 mg/L or higher, over a 40% increase in the risk of hypertension was found. This is independent of any other risk factor such as obesity, hyperlipidemia or diabetes.

Of course, the presence of a CRP of less than one does not assure that hypertension will not develop from one of the other factors (such as obesity), only that once the study is controlled for these factors there is no associated increased risk of hypertension with these low CRP levels.

Inflammation appears to play a role in the development of atherosclerosis with subsequent myocardial infarction and stroke. This is the first study to link inflammation and hypertension. It is postulated that CRP decreases production of nitric oxide by endothelial cells that may indirectly promote vasoconstriction, leukocyte adherence, platelet activation, oxidation, and thrombosis.

Despite these findings, it does not seem reasonable to consider adding C-reactive protein to screening labs for life insurance at this time purely for the purpose of trying to predict who will develop hypertension. This is a topic, however, that deserves monitoring for future developments.