C-Reactive Protein and Blood Pressure in the Acute Phase After an Ischemic Stroke

To the Editor:

Elevated blood pressure levels have been associated with an increased risk of stroke and of cardiovascular disease. It is now well established that vascular inflammation is an independent risk factor for the development of atherosclerosis. Furthermore, low grade of inflammation, assessed by C-reactive protein (CRP), significantly predict the risk of future ischemic stroke. Thus, the mechanism underlying the link between elevated blood pressure and an increased risk of stroke may be inflammation. Engström et al demonstrated in a recent Stroke article that increased levels inflammation-sensitive plasma proteins are associated with elevated blood pressure and these proteins are associated with an increased risk of stroke in patient with high blood pressure. From this point of view, we have the possibility, using the database of the Villa Pini Stroke Data Bank to extend their observations regarding the relationship between CRP and blood pressure levels in acute ischemic stroke. Previous data demonstrated that ischemic stroke triggers an acute phase response resulting in a rise of circulating CRP level. However, the amount of the inflammatory response to ischemic stroke is variable: about 25% of patients with first-ever ischemic stroke have normal level of CRP after stroke implying that ischemic stroke itself does not induce a full-blown acute phase response. CRP elevation can result from a variable intensity of the individual acute phase response to cerebral ischemia but it is not known if blood pressure levels in the acute phase after stroke can influence levels of inflammation markers. To verify this hypothesis we analyzed the relationship between systolic diastolic blood pressure and CRP levels within 24 hours after stroke onset in 507 ischemic stroke patients included in the recruitment period between March 1998 to March 2000.

At the entry, mean systolic blood pressure was 160±16 mm Hg and mean diastolic blood pressure was 94±13 mm Hg. Of course, patients with a history of arterial hypertension had a significantly higher mean systolic (164±8 versus 148±24 mm Hg; P<0.0001, Student’s t test) and diastolic blood pressure (96±8 versus 86±19 mm Hg; P<0.0001) than patients without. Blood pressure decreased on average by 11 mm Hg in systolic and 14 mm Hg in diastolic during in-hospital stay.

Log-normalized concentration of CRP within 24 hours after stroke was significantly correlated with both systolic (r=0.47; P<0.0001) and diastolic (r=0.50; P<0.0001) blood pressure at the entry, but modestly. Patients without a history of arterial hypertension had significantly higher levels of CRP at the entry than patients with a documented history (2.0 [1.0 to 3.4] versus 1.0 [0.5 to 2.75] mg/dL; P<0.0001, Mann-Whitney U test). However, significantly higher median levels of CRP were found in patients with higher levels of both systolic or diastolic blood pressure irrespective to the history of arterial hypertension.

The association between blood pressure and the odds of having an elevated CRP level (≥1.5 mg/dL) was assessed by logistic regression analysis. In a model that adjusted for systolic blood pressure, history of arterial hypertension, diabetes mellitus, coronary heart disease, demographic factors, cholesterol, measures of obesity, smoking, alcohol abuse, stroke severity, neurological findings, and antihypertensive medication use, a 10 mm Hg increase in systolic blood pressure was associated with a 39% increase in the odds of having an elevated CRP level (odds ratio [OR]=1.39, 95% confidence intervals [CI] 1.21 to 1.61, P<0.0001). When the same model was re-run adjusted for diastolic blood pressure instead of systolic blood pressure, a 10 mm Hg rise in diastolic blood pressure was associated with a significant 42% increase in the odds of having an elevated CRP level (OR=1.42, 95% CI 1.17 to 1.73, P=0.0004).

In conclusion, our preliminary results suggest that elevated levels of systolic or diastolic blood pressure in the acute phase after an ischemic stroke are associated with elevated CRP levels. An acute increase of blood pressure more than a history of arterial hypertension determine higher levels of CRP after stroke. Probably, the levels of blood pressure after an ischemic stroke are one of underlying processes related to inflammation and they are relevant in the inflammatory response after an ischemic stroke. These data confirm the recent observation by Engström and colleagues that the high pressure values are associated with the production of inflammation-sensitive plasma protein. Many speculative hypotheses could be made regarding the relationship between blood pressure and the inflammatory mechanism in ischemic stroke. From this point of view, because higher CRP levels are an independent prognostic factor after stroke and, apparently, high blood pressure increases CRP levels, the current idea on the acute hypertension and its treatment after stroke probably should be revisited.

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