Is C-reactive protein an independent risk factor for essential hypertension?

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We read with interest the paper by Bautista and colleagues [1], recently published in a previous issue of the Journal. Over a population of 300 subjects aged 30 years or more, they found that prevalence of hypertension significantly parallels C-reactive protein (CRP) serum levels, suggesting that inflammation may be a risk factor in developing essential hypertension. We want to contribute to this hypothesis with our experience. In a population of 89 hypertensive elderly patients (mean age 75.1 ± 8.5) consecutively admitted to a Geriatric Unit, we studied the association between CRP serum levels and the circadian blood pressure (BP) profile using 24 h non-invasive BP monitoring (Takeda TM 24-30; A&D Instrument Ltd, Abingdon, UK). Exclusion criteria were the presence of diabetes, coronary heart disease or acute inflammatory events (CRP serum level higher than 1.50 mg/ml). On the basis of previous large epidemiological evidence [2], we defined non-dipper subjects as those with a night-to-day ratio of 100% or higher, and dippers those with a lower night-to-day ratio. Firstly, we analyzed CRP serum levels, finding that they were significantly different between dippers and non-dippers (1.0 ± 0.8 and 0.7 ± 0.5 mg/dl, respectively; P = 0.02). We then stratified three groups of patients accordingly, to CRP serum levels (0.1–0.69, 0.70–1.10 and 1.11–1.50 mg/dl), observing that the proportion of non-dippers increased from 22 to 33 to 45%, with rising CRP serum levels. With the lowest CRP level group taken as the reference, the crude odds ratio of the association were 2.0 [95% confidence interval (CI) 0.6–6.7] and 3.1 (95% CI, 1.0–10.0) in the intermediate and third group, respectively,

\[ P_{\text{for trend}} = 0.04. \]

Our results are in line with the report of Bautista and colleagues [2,3] in the association between inflammation and hypertension, since non-dipping BP status is a common finding in essential hypertension with advanced target-organ damage [3]. Furthermore, because both inflammation and loss of night-time BP fall are associated with higher cardiovascular and cerebrovascular morbidity [4,5], we suppose that patients with co-occurrence of these two conditions may be at greater risk of disease severity. We are planning the follow-up of our patients to test this hypothesis.

References
