To the Editor:

We read with great interest the article by Ginsberg et al concerning the safety of albumin therapy for acute ischemic stroke. The authors found that albumin doses ranging from 1.37 to 2.05 g/kg were generally well tolerated by stroke patients, and that the main adverse event, a mild-moderate pulmonary edema, was infrequent and easily manageable. The main physiological consequences of albumin administration were: (a) an elevation of albumin plasma levels, (b) a dose-dependent hematocrit reduction, and (c) an increase in B-type natriuretic peptide (BNP) levels which, however, was not related to the appearance of cardiac adverse events.

The latter observation by Ginsberg et al is in agreement with the results of a cross-sectional epidemiological study in a general elderly population, by which we showed the existence of an inverse correlation between hematocrit and the N-terminal of BNP precursor (NT-proBNP), independent of numerous other clinical and hematopoietic variables, as well as of the principal echocardiographic mass and volume indexes of cardiac chambers. The relationship was rather strong and was able to explain why BNP levels are higher in women than in men. The cross-sectional nature of our study allowed the detection of the inverse association between hematocrit and NT-proBNP but not the demonstration of the cause-effect relationship. We speculated that a low hematocrit may represent a direct stimulus to the production of BNP, through mechanisms and receptors that are presently unknown, within a homeostatic process of hemodilution control. Subsequently, the higher BNP levels, by increasing sodium excretion and diuresis, would tend to bring hematocrit toward normality. The study by Ginsberg et al, in which the active administration of albumin caused a lowering of hematocrit with BNP increase following, has shown the direction of the causal flow. In addition, our epidemiological data help to confirm the substantial safety of albumin administration to stroke patients.

Disclosures

None.

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Albumin-Induced Hematocrit-Lowering and B-type Natriuretic Peptide Increase
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