Role of Hypertension in the Development of Cerebral Atrophy in Uremia

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Dear Sir,

The etiopathogenetic role of hypertension in causing cerebral atrophy (CA) in uremic and hemodialyzed patients has been extensively studied by our group since 1981, when occasionally cerebral computed tomography (CCT) evidenced CA in young patients under regular hemodialysis treatment (RHT).

Our first attempt to draw etiopathogenetic conclusions concerning CA in RHT patients appeared in 1985 [1]: in this study on 30 patients, a statistical elaboration by discriminant analysis of variables indicated a decreasing correlation of CA with blood pressure and oral Al(OH)₃ intake, while blood cholesterol levels, vascular calcifications, and blood triglyceride levels were less correlated. No substantial achievement was obtained by doubling the number of cases [2]. Only now, in a much larger group of patients, is there more convincing evidence that hypertension plays a principal role in the pathogenesis of CA [3].

In any case, the difficulties of drawing definitive conclusions are due to the fact that although all our patients were on conservative treatment or on RHT [4] and were < 55 years old (an age at which CA attributable to cerebral involution would be considered abnormal), they were not homogeneous as regards Al(OH)₃, dyslipidemia, cardiopathy, or secondary hyperparathyroidism. We would like to emphasize the objective difficulty in comparing CCT scans made with first- and third-generation scanners. Interpretation of the most recent scans was facilitated by region of interest and segmentation analyses. In another study [4] we were extremely careful to abstain from any etiopathogenetic conclusions, on account of the limited number of cases and because some of our patients with CA, in the past, had taken considerable amounts of Al(OH)₃, but we agree with the colleagues and their analysis of the correlations between CA and hypertension.

In a recent study [5], we found periventricular white matter lucencies in 6.9% of our population on RHT. Periventricular leukoencephalopathy has probably been underestimated in this study due to the lesser sensitivity of the CCT in comparison to T2-weighted magnetic resonance images. These hypertensive lesions in deep and subcortical white matter are incidental findings in neu-rologically asymptomatic patients, and incidence and severity of
the lesions are higher in hypertensive non-uremic patients, even when they are on adequate antihypertensive treatment [6]. Vascular alterations of small vessels and arteriolar border zones may lead to chronic ischemic states [7].

To conclude, we believe that although hypertension plays a significant role in the formation of leukoaraiosis, other factors are involved in the development of CA in uremia [8,9].

References


