Blood Amino Acid Levels and Erythropoietin Treatment in Hemodialysis Patients

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Dear Sir,
Chronic renal failure (CRF) promotes a number of abnormalities in amino acid (AA) metabolism, which are reflected by changes in circulating levels of AA [1]. Changes in blood AA profile, similar to those observed in protein-calorie malnutrition, occur in patients with relatively preserved renal function under high protein and calorie intake and are therefore caused by renal failure per se. Studies carried out to evaluate AA exchange across the organs which pay a major role in AA metabolism show that blood levels of many AA are the consequence of altered kidney or muscle metabolism. At the same time, abnormalities in blood AA may affect AA uptake by the brain and hepatosplanchic bed [2]. In advanced uremia a low calorie/protein intake can aggravate blood AA profile, since in this condition changes in blood AA profile mimic those observed in CRF patients. Abnormalities persist in hemodialysis patients, even if they are adequately dialyzed and the protein intake is sufficient [3]. Such abnormalities may, in association with the catabolic stress of hemodialysis, increase protein requirements and expose patients to malnutrition.

A previous report by Riedel et al. [4] showed that correction of anemia by treatment with rhEpo could significantly improve, over a short-term period, many abnormalities in circulating AA levels in hemodialysis patients. In a second report [5] the same authors compared plasma amino and α-keto acid concentrations in hemodialysis patients with modest (Hb 12.9 ± 1.2 g/dl) or severe (Hb 7.1 ± 2.1 g/dl) anemia; alterations in AA levels were much more marked in severely anemic patients. The authors concluded that ‘severity of anemia influences pattern of AA and α-keto acids in hemodialysis patients’. Unfortunately, both studies did not report data on the nutritional status of patients as well as on their protein and calorie intake. The original report by Riedel et al. [4] prompted us to evaluate the long-term effects of correction of anemia by rhEpo treatment on blood AA in hemodialysis patients [6]. As the blood AA profile in
malnutrition mimics that observed in CRF, patients without evidence of protein malnutrition were selected for this study. Patients had severe anemia with pretreatment hemoglobin values which were only slightly higher (+13%) than in patients studied by Riedel et al. [4] (7.0±0.8 vs. 6.2±1.5 g/dl). The degree of correction of anemia (and oxygen availability) was similar in both studies [up to 10 g/dl, +43%, in our study; up to 9.5 g/dl, +53%, in ref. 4]. However, in contrast to Riedel et al. [4], we were not able to detect any change in AA levels, whose alterations persisted unmodified for the entire 12-month study. We conclude that abnormalities in circulating AA observed in hemodialysis patients without protein malnutrition are not modified by correcting anemia with rhEpo. Obviously, it cannot be excluded that the correction of anemia by the associated sensation of well-being and increased food intake can actually improve the nutritional status in hemodialysis patients with exogenous malnutrition.

References


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