Dear Sir,

The anemia of chronic renal insufficiency is the result of diminished bone marrow production due to the lack of renal erythropoietin and the reduced survival of red blood cells. In vitro, lack of carnitine is responsible for an increase of fragility of normal erythrocytes [1]. Nevertheless publications tend to suggest that hematocrit improves after the treatment with L-carnitine in hemodialyzed patients with a low circulating level of carnitine [2-6]. The acidified glycerol lysis test (AGL test), classically used to evaluate constitutional erythrocyte membrane fragility [7, 8], can be performed with only 150-200 µl of blood remaining after the routine blood count has been done. In these conditions, we carried out AGL tests on 33 hemodialyzed patients (18 males), aged 20-81: 24/33 (72%) had a positive result.

Following the recommendations of Wanner and Hörl [9], we routinely survey carnitine levels [with spectrophotometric method, 10] in our patients and treat those with free carnitine lower than 30 µmol/l with L-carnitine mg/kg intravenously at the end of each dialysis session. Among the 33 patients tested with the AGL test, 14 showed a low carnitine level and 10/14 had a positive AGL test. After months of treatment with L-carnitine, the 4 negative patients remained negative by the AGL test and 6 of the positive patients became negative. This result, analyzed by Mac Nemar paired χ², is statistically significant (p < 0.05).

Because circulating levels of carnitine decrease only after depletion of tissue stores [9], and because a free carnitine level higher than 30 µmol/l is insufficient to prove the absence of deficiency, we also treated 4 patients with carnitine levels higher than 30 µmol/l who had a positive AGL test. Two became negative after treatment. Therefore, independently of the carnitine level, out of 18 patients with a positive AGL test, 12 became negative after therapy (66%: p < 0.02).

Among the 18 patients treated, 8 received erythropoietin. With a χ² test, we demonstrate that the ratio of patients responsive to carnitine treatment does not differ significantly from that treated with erythropoietin (5 out of 8) and from patients without this therapy (7 out of 10). Concerning the 8 patients receiving erythropoietin, this treatment was decreased or stopped in 4 of them
without significant modification of hematocrit or hemoglobin level, and was maintained or increased in other patients.

These preliminary results tend to demonstrate the therapeutic effect of L-carnitine in improving membrane fragility of erythrocytes in dialyzed patients without erythropoietin involvement. We propose that a reduction of doses and frequency of therapy with erythropoietin may be achieved by addition of a low dose of L-carnitine. Our results based on a small population need to be proved in a larger study.

References


