Transcobalamin II. A Specific Marker of Renal Involvement in Essential Mixed Cryoglobulinemia

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Dear Sir,

Essential mixed cryoglobulinemia (EMC) is an uncommon disease characterized by weakness, arthralgias, purpura and mixed IgM-IgG cryoglobulinemia [1]; in addition, about half of the cases develop glomerulonephritis which might appear as an initial or evolutionary manifestation of the disease[1]. Prognosis of EMC largely depends on the possible occurrence of renal involvement, which, on the other hand, may respond to treatment with corticosteroids and immunosuppressive drugs and/or plasmapheresis [2–4]. In EMC there is no parameter, not even cryocrit [5], that correlates with either the activity of the disease or with renal involvement.

Transcobalamin II (TC II) is a serum carrier protein of vitamin B12. In the last few years it has been proved by several authors [6, 7] that patients with systemic lupus erythematosus (SLE) show increased levels of TC II, there being a good correlation between TC II levels and activity of SLE. While performing a prospective study on the behavior of TC II in different systemic diseases, we verified an increase on TC II levels in patients who had EMC with glomerulonephritis. Furthermore, we observed that levels of TC II and activity of renal involvement were closely correlated.

Seven patients with EMC were evaluated. The diagnosis of EMC was established on the basis of the presence of typical symptoms and the exclusion of any infectious, neoplastic or autoimmune disease. Two cases had membranoproliferative glomerulonephritis as determined by histological methods. These two patients were treated with corticosteroids and cyclophosphamide, a biological remission of renal involvement being proved subsequently. As control groups we used 95 healthy donors and 27 patients with chronic renal failure on an hemodi-alysis program. The age of controls was similar to that of patients with EMC. Assessments of TC II were made by means of radioimmunoassay (Dircosa TC II kits). Ten TC II assessments were performed on patients with EMC and renal involvement, 6 while they had active renal involvement (ARI; histological and biological) and 4 during the period of inactive renal involvement (IR; biological). Eleven assessments of TC II were performed on the 5 EMC patients without renal involvement.
The level of TC II in the healthy control groups was 1.206 ± 308 ng/l (x- ± SD). The chronic-renal-failure group had 1.458 ± 524 ng/l. The level of TC II in EMC patients with renal involvement was 2.318 ± 661 ng/l during the period of ARI and 1.242 ± 452 ng/l during IRI. In the EMC group without renal involvement, the level of TC II was 1.426 ± 336 ng/l. There is a statistically significant difference (p < 0.05) between the EMC group with ARI and every other group.

TC II increased in those patients with EMC and ARI, returning to normal levels as patients responded to treatment. Moreover, TC II levels were normal in those EMC patients who didn’t have renal involvement but who did have other organ systems involvements such as hepatic or neurological. Thus, the behavior of TC II was that of a specific marker for active glomerulonephritis. The reason why TC II increases in patients with EMC and ARI remains unknown to us. A possible mechanism would be a dysfunction of the reticuloendothelial system. This has been suggested as a mechanism in order to explain increases of TC II [7] and it is known to occur in patients suffering from EMC with renal involvement, but not in those without renal involvement [8].

Obviously ours is a short series and requires corroborative studies. Nevertheless, in the light of our own results, we think TC II could be a good biological parameter indicative of renal involvement in EMC as well as a useful monitor of its response to treatment.

References

Errata
In the article by Struijk et al., published in Nephron, Vol. 44, p. 384 (1986), the title ‘Patient Viral Peritonitis in a Continuous Ambulatory Peritoneal Dialysis’ should be corrected and read ‘Viral Peritonitis in a Continuous Ambulatory Peritoneal Dialysis Patient’.

In Nephron
Vol. 44
No. 4 (1986)
on pp. 295
355
379 and 385
the reference line and CCC-Code should be corrected to 1986 not 1987 as erroneously printed. On p. 356 figure la and figure lb are transposed: figure la should be figure lb and vice versa.