Biological Markers of Inflammation and Carpal Tunnel Syndrome in Dialysis Patients

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Fig. 1. Acute-phase reactants in the three study groups. Data are mean ± SD. ES = Group with carpal tunnel syndrome; ■ = control group; □ = normal subjects. α-1-Antitrypsin. β-2-Macroglobulin. c C-reactive protein. d α1-Acid glycoprotein.

Blood sampling was performed immediately before dialysis. Acute-phase reactants were measured by using a commercially available nephelometric method (Ben-ring). To evaluate the biological variability of these measurements, blood sampling was repeated in all cases after an interval of 1 month.

As shown in figure 1, α1-Antitrypsin and β-2-Macroglobulin were significantly higher in carpal tunnel patients than in the control group. C-reactive protein showed a similar tendency (p = 0.14). α1-Acid glycoprotein was increased by the same extent in the two dialysis groups in comparison with healthy subjects.

Dear Sir,

Carpal tunnel syndrome is a frequent and disabling complication of long-term dialysis. This syndrome is considered secondary to the deposition of β2-microglobulin in the carpal tunnel synovia and tendons, a process which is favored by the retention of this substance. β2-Microglobulin accumulation may depend on various factors such as poor dialysis removal, enhanced generation rate promoted by subclinical inflammatory processes [1] and/or reduced metabolic degradation.

The study of acute-phase reactants in carpal tunnel syndrome is of interest because some of these substances, in addition of being mediators of inflammation, display antiproteolytic activity and in this way may reduce β2-microglobulin catabolism. We, therefore, compared the plasma levels of 4 well-characterized acute-phase reactants (α1-Antitrypsin, β2-Macroglobulin, α1-Acid glycoprotein and C-reactive protein) in dialysis patients with and without this syndrome.

Nine hemodialysis patients with well-documented carpal tunnel syndrome [abnormal electromyographic (EMG) studies and/or clinical improvement after surgical decompression] and 9 hemodialysis patients without carpal tunnel syndrome (no clinical symptoms and negative
EMG studies) participated in the study. The two groups were well matched for duration of dialysis treatment (12 ± 2 vs. 13 ± 2 years). All had been dialyzed with cuprophane membranes. At the time of the study no patient in either group had clinical evidence of inflammatory processes and all were virtually anuric.

On second testing, α₁-antitrypsin and α₂-macroglobulin showed very little variation in patients affected by carpal tunnel syndrome, remaining consistently raised (coefficients of variation = 17% and 14%, respectively), while all patients remained free of obvious inflammatory processes. The accumulation of α₁-antitrypsin and α₂-macroglobulin and C-reactive protein in patients with carpal tunnel syndrome is in keeping with the idea that these patients harbor subclinical inflammations. α₂-Acid glycoprotein retention seems to be a nonspecific phenomenon mainly related to the uremic state. In theory, accumulation of α₁-antitrypsin and α₂-macroglobulin may participate in the pathogenesis of carpal tunnel syndrome by their ability to inhibit proteolytic processes. In this regard, it is worth mentioning that a link between antiproteasic activity and α₂-microglobulin has been reported in hemodialysis-associated amyloidosis [2]. On the other hand, it is also possible that inflammation per se enhances β₂-microglobulin production and favors its deposition in tissues [1].

Our data suggest that acute-phase reactants participate in the pathogenesis of carpal tunnel syndrome. Further studies are required to characterize their role in hemodialysis amyloidosis.

References