Are muscle cramps in Isaacs' syndrome triggered by human immunoglobulin?

Ishi A et al reported the clinical evaluation of plasma exchange and treatment with high dose intravenous immunoglobulin (IVIg) in a patient with Isaacs' syndrome. The rationale for either treatment in this patient was a possible autoimmune aetiology. The differential treatment response was remarkable: after plasma exchange the symptoms of continuous muscle activity almost disappeared whereas after IVIg treatment muscle cramps gradually increased. The authors state that the reason for this divergence is unclear, and suggest that IVIg may have a similar adverse effect in Isaacs' syndrome as has recently been reported in patients with Guillain-Barré syndrome.

We would like to draw attention to another explanation for the differential treatment response of plasma exchange and IVIg, and propose the possibility of a direct effect of IVIg on muscle cells, causing muscle cramps in the patient with Isaacs' syndrome. By applying IgG antibodies by IVIg administration, the decrease in the nerve terminal may lead to a decrease in the nerve terminal. Our report is the first study of the use of IVIg in Isaacs' syndrome, and thus we cannot really assess the effectiveness of this treatment. There is, however, one patient with Isaacs' syndrome who improved with IVIg treatment (Wintzen et al and A R Wintzen (unpublished communication)). It would seem, therefore, that the effect of IVIg may be dependent on the specifics of each case. There is likewise the possibility that the effect may be altered by the type or dose of human immunoglobulin.

Complement alterations in the CSF of patients with myasthenic syndrome

Recently, Taubö and Yamada reported increased CSF concentration of C4d and increased C4d index values in patients with myasthenic syndrome and suggested that this finding may be due to complement activation that could play a part in motor neuron degeneration. Since 1985, we have found high levels of C3c but not changes in C3c index values and other complement fractions in CSF from patients with myasthenic syndrome correlating with C1q and albumin and, more specifically, with the CSF protein concentrations. We proposed that the increase in C3c fraction could be due in part to leakage through the damaged blood-brain barrier but also to decreased binding to specific complement receptors on CNS lymphocytes that leads to complement depletion, thus reducing tissue damage. This interpretation focuses on the biochemical and functional changes in cell membranes from patients with myasthenic syndrome. The role of the immunological alternations in myasthenic syndrome pathogenesis needs further investigation.

Somatization in neurological practice

I was interested to read the article by Ron on somatization in neurological practice. The inability to make a specific diagnosis in neurological outpatient practice is something that I referred to in a paper published in this Journal in 1995. An analysis of 7838 successive new referrals to my clinics established that some 26-5% did not have a specific diagnosis, even in some cases after extensive investigation. Ron might be interested to know that among the same number of patients 29% or 3-8% had some evidence of conversion hysteria. Based on an earlier study, also published, one would have expected probably 50% of these...