

# Myelopathy in Sjögren's Syndrome

## A Causative Role for Cobalamin (Vitamin B<sub>12</sub>) Deficiency

As Rogers et al. report,<sup>[1]</sup> the pathogenesis of CNS Sjögren's syndrome, including myelopathy, is not well understood. Several causative factors have been implicated, but they are often not well documented. However, in clinical practice, it is important to keep in mind that several CNS manifestations associated with Sjögren's syndrome may be related to cobalamin (vitamin B<sub>12</sub>) deficiency, as we have previously demonstrated.<sup>[2,3]</sup> Here we report additional data.

We have studied the cobalamin status and the neuropsychiatric manifestations in 80 patients with Sjögren's syndrome.<sup>[4]</sup> These patients were recruited from an observational cohort study that involved >100 patients with primary Sjögren's syndrome followed at the University Hospital of Strasbourg, France. The median age of the patients was 55.8 years and 81.5% were women. For all patients, the mean serum vitamin B<sub>12</sub> level (Vitamine B<sub>12</sub>, EIA, Abbott, Rungis, France) was  $419 \pm 331$  pg/mL. Seven patients (8.8%) had 'established' vitamin deficiency with serum vitamin B<sub>12</sub> levels <200 pg/mL. Twelve (15%) and 33 (41.2%) of the patients had serum vitamin B<sub>12</sub> levels between 200 and 250 pg/mL, and between 250 and 300 pg/mL, respectively, and are thus categorised as 'possible' and 'potential' vitamin B<sub>12</sub> deficiencies. The prevalence of vitamin B<sub>12</sub> deficiency was 5.3% in our Department of Internal Medicine over the same period.<sup>[5]</sup> Neuropsychiatric manifestations were noted in 11% of all the patients. Central neurological and psychiatric manifestations (optic neuritis, impairment of mental status, dysesthesias of the hemi-body related to myelopathy) were noted in 3 of 19 patients (15.8%) with 'established' and 'possible' vitamin B<sub>12</sub> deficiencies (serum vitamin B<sub>12</sub> levels <250 pg/mL) versus 3 of 61 (4.9%) patients for the remainder of this cohort.

These preliminary results suggest that vitamin B<sub>12</sub> deficiency may be more frequent in patients with primary Sjögren's syndrome than previously thought. Thus, in our opinion, in practice it is advisable to exclude the possibility of cobalamin deficiency in patients with neuropsychiatric symptoms, including myelopathy, before considering a possible involvement of Sjögren's syndrome. In this situation, oral or parenteral cobalamin therapy may be useful, as we have demonstrated.<sup>[2-4]</sup>

Emmanuel Andrès,<sup>1</sup> Laure Fédérici<sup>1</sup> and Jean Sibilia<sup>2</sup>

1 Department of Internal Medicine, Clinique Médicale B, University Hospital of Strasbourg, Strasbourg, France

2 Department of Rheumatology, University Hospital of Strasbourg, Strasbourg, France

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Correspondence and offprints: Dr Emmanuel Andrès, Service de Médecine Interne, Diabète et Maladies Métaboliques, Clinique Médicale B, Hôpital Civil, Hôpitaux Universitaires de Strasbourg, 1 place de l'Hôpital, Strasbourg, 67 091 Cedex, France.

E-mail: emmanuel.ANDRES@chru-strasbourg.fr