Thoracic Spinal Cord Atrophy Secondary to Vitamin B12 Deficiency

Frederico Carvalho de Medeiros¹, Lucas Alverne Freitas de Albuquerque²* and Cíntia Alvarenga Pereira¹

¹Department of Neurology, Santa Casa de Belo Horizonte Hospital, Minas Gerais, Brazil
²Department of Neurosurgery, Santa Casa de Belo Horizonte Hospital, Minas Gerais, Brazil.

*Corresponding author: Lucas Alverne Freitas de Albuquerque, Rua Padre Rolim, number 492, apt 101. Zip code 30130-090. Santa Efigênia, Belo Horizonte – MG, Brazil, Tel: +55(85)96279966; Email: lucasalverne@yahoo.com.br

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Description

A 57 year-old woman searched for a neurological Service for the first time when she was 45-year-old, complaining about weakness in lower limbs for ten months period. The physical examination reported was of sensitive level at T5, reduced proprioception, Romberg’s sign and ataxic gait. A Work up discovered a low Vitamin B12 level of 64pg/ml. Vitamin B12 reposition was initiated, however the patient failed to follow up.

The patient looked for our Hospital with 57-year-old moving herself using a walker. A cervical and thoracic spinal cord MRI was performed (Figure 1). A sagittal and axial thoracic spinal cord MRI showed diffuse spinal cord atrophy with preservation of signal intensity at T2-weighted image (T2WI).

The myelopathy of vitamin B12 deficiency is rare and normally affect the lower cervical and upper thoracic regions [1,2]. Extensive thoracic myelopathy due to vitamin B12 deficiency is very rare [2]. The predominant MRI finding is demyelination involving the dorsal columns, showed by the hyperintense signal at T2 [1-3]. In these cases the clinical and radiological improvements are possible if early therapy is initiated promptly [1].

Spinal cord atrophy secondary to prolonged vitamin B12 deficiency is an extremely rare complication of the myelopathy. We believe that

Figure 1: Sagittal T2-weighted MRI shows: A) preserved cervical spinal cord and B) important thoracic spinal cord atrophy; C) axial T2-weighted MRI demonstrates a pronounced reduction in the diameter of the thoracic spinal cord.

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Spinal cord atrophy secondary to prolonged vitamin B12 deficiency is an extremely rare complication of the myelopathy. We believe that
delayed treatment and prolonged inadequate therapy induces irreversible axonal degeneration with progressive fibrous gliosis, promoting spinal cord atrophy (Figure 1) [1,3]. In this case it was associated to a very poor response to vitamin B12 replacement.

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References