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Letter with reference to the article "Importance of electromyographic studies in the diagnosis of orthostatic tremor"[☆]

Carta en relación con el artículo «Importancia del estudio electromiográfico en el diagnóstico del temblor ortostático»

Sir,

We read with great interest the article published recently by Yagüe et al.¹ about the importance of electromyographic study in the diagnosis of orthostatic tremor. Our intention is not to assess the pathophysiological-diagnostic aspect of the article, brilliantly exposed by the authors who provide electromyographic concepts that demonstrate the growing and undeniable importance of electrophysiological analysis in the study of movement disorders. As the authors note, orthostatic tremor is an idiopathic motor disorder, although its possible association with pons lesions² or cerebellar degeneration^{3,4} has been reported. Both of these constitute symptomatic forms of orthostatic tremor. It has been speculated that these anatomical structures (brainstem and cerebellum) could be directly involved in the pathogenesis and modulation of orthostatic tremor,^{5,6} in a manner similar to what might happen in essential tremor.⁷⁻⁹ We have noted that, in the case presented by Yagüe et al.,¹ the patient presented a deficiency of vitamin B12 in her medical history, and this has motivated us to write this letter to highlight the possible association existing between orthostatic tremor and vitamin B12 deficiency. Eleven years ago, our group published the case of a patient with orthostatic tremor who also suffered vitamin B12 deficiency.¹⁰ While we recognise that this association is difficult to justify from the standpoint of biological plausibility, the fact that, following the reinstatement of the deficient vitamin, the tremor disappeared does strengthen this causal association. Since then, other cases have been published in which vitamin B12 deficiency has been associated with tremor.^{11,12} Furthermore, several neuroimaging studies have revealed abnormalities in the cerebellum and pons of patients with vitamin B12 deficiency.^{13,14} We believe these studies lend plausibility to the hypothesis of there being a causal inference between vitamin B12 deficiency and orthostatic tremor.

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Importance of electromyographic studies in the diagnosis of orthostatic tremor. Reply[☆]

Importancia del estudio electromiográfico en el diagnóstico del temblor ortostático. Réplica

Sir,

We appreciate the letter by Benito et al. emphasising the possible association between orthostatic tremor and vitamin B12 deficiency, which highlights the causal association between these 2 entities and the existing literature on this subject.^{1–6} Indeed, being aware of this association, and given the medical history of the patient, we did review her vitamin B12 levels. At the time they were between 249 and 421 pmol/l (132–857) and mean corpuscular volume was around 85 fL (80–96) in successive analyses conducted between 2006 and 2009, when we first started treating her.

The patient had a history of gastric neoplasm in 2006 that required total gastrectomy and cholecystectomy, as well as monthly intramuscular supplements of vitamin B12, from 2006 until the present time. However, the clinical onset of instability in the lower limbs took place in 1996, 10 years before the history of gastric neoplasm and subsequent gastrectomy that caused the vitamin B12 deficit.

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Bilateral carotid occlusion and progressive stenosis of vertebral arteries after radiotherapy in a young patient[☆]

Oclusión carotídea bilateral y estenosis progresiva de arterias vertebrales posradioterapia en paciente joven

Sir,

An increased risk of stroke has been reported in patients under 60 who have received radiation therapy (RT) for head and neck neoplasms, due to the fact that irradiation of these regions causes accelerated atherosclerosis, enhanced by the coexistence of vascular risk factors. The onset of cerebral ischemia symptoms usually occurs 10 years after radiation

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Nevertheless, it is important to bear in mind this association and we wish to thank Benito et al. for highlighting this potential causal inference, given the reversibility of orthostatic tremor after treatment and replacement of vitamin B12 levels.

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therapy,¹ although the exact course of post-RT arteriopathy and the most adequate treatment have not been properly established.

We report the case of a young stroke patient in relation to a major, progressive post-RT arteriopathy of extracranial cerebral arteries.

The patient was a 45-year-old male, a heavy ex-smoker with a history of nasopharyngeal carcinoma treated with RT at age 25. The total dose of RT received by the patient was 70 Gray (Gy), at a rate of 2 Gy daily. The irradiated area encompassed the nasopharynx (area of primary tumour) and lymph node chains (cervical region). He came to the emergency service due to a loss of strength in the left side of the body and dysarthria upon awakening. In the previous 2 days, he had presented self-limited episodes of neurological symptoms consisting of tongue paresthesia and a feeling of dizziness. We performed cranial CT scans, an electroencephalogram, chest radiography and laboratory studies, which all resulted normal. The patient was admitted at a stroke unit, where an MRI revealed an acute ischemic lesion in the right fronto-temporal region (Fig. 1A). The transcranial Doppler (TCD) showed a very significant acceleration of the right middle cerebral artery