systemic symptoms; this requires a high level of clinical suspicion. Early diagnosis enables the provision of effective treatment able to resolve symptoms and even to prevent lesions from spreading to other organs or regions of the nervous system; Whipple disease is potentially fatal if the patient does not receive targeted treatment.

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Improved functional capacity in Huntington disease after Nordic walking training: A case report☆

Mejoría de la capacidad funcional en la enfermedad de Huntington tras un programa de marcha nórdica. A propósito de un caso

Dear Editor:

Huntington disease (HD), the most frequent genetically determined type of chorea, is an autosomal dominant, progressive neurodegenerative disorder that causes physical, cognitive, and psychological impairment.[1] Physical exercise is considered a useful rehabilitation therapy in HD, although it is yet to be determined what type provides the greatest potential benefits in slowing progression of the disease.[2] Nordic walking may be an interesting physical therapy in HD due to the low level of fitness required, the simplicity of the exercise, and the reported effects on independence and motor symptoms in patients presenting neurodegenerative symptoms.[3] We describe the case of a patient with early-intermediate stage HD who presented changes in functional status after participating in a Nordic walking programme.

Our patient was a 59-year-old woman who consulted due to gait and balance alterations associated with rapid, involuntary dyskinetic movements. Diagnosis of early-intermediate stage HD was confirmed after a genetic study revealed an abnormal CAG trinucleotide repeat expansion in *IT15*. Symptomatic treatment was started, and improved motor control. Two months after the diagnosis, and after visiting the Faculty of Education and Sports Sciences at the

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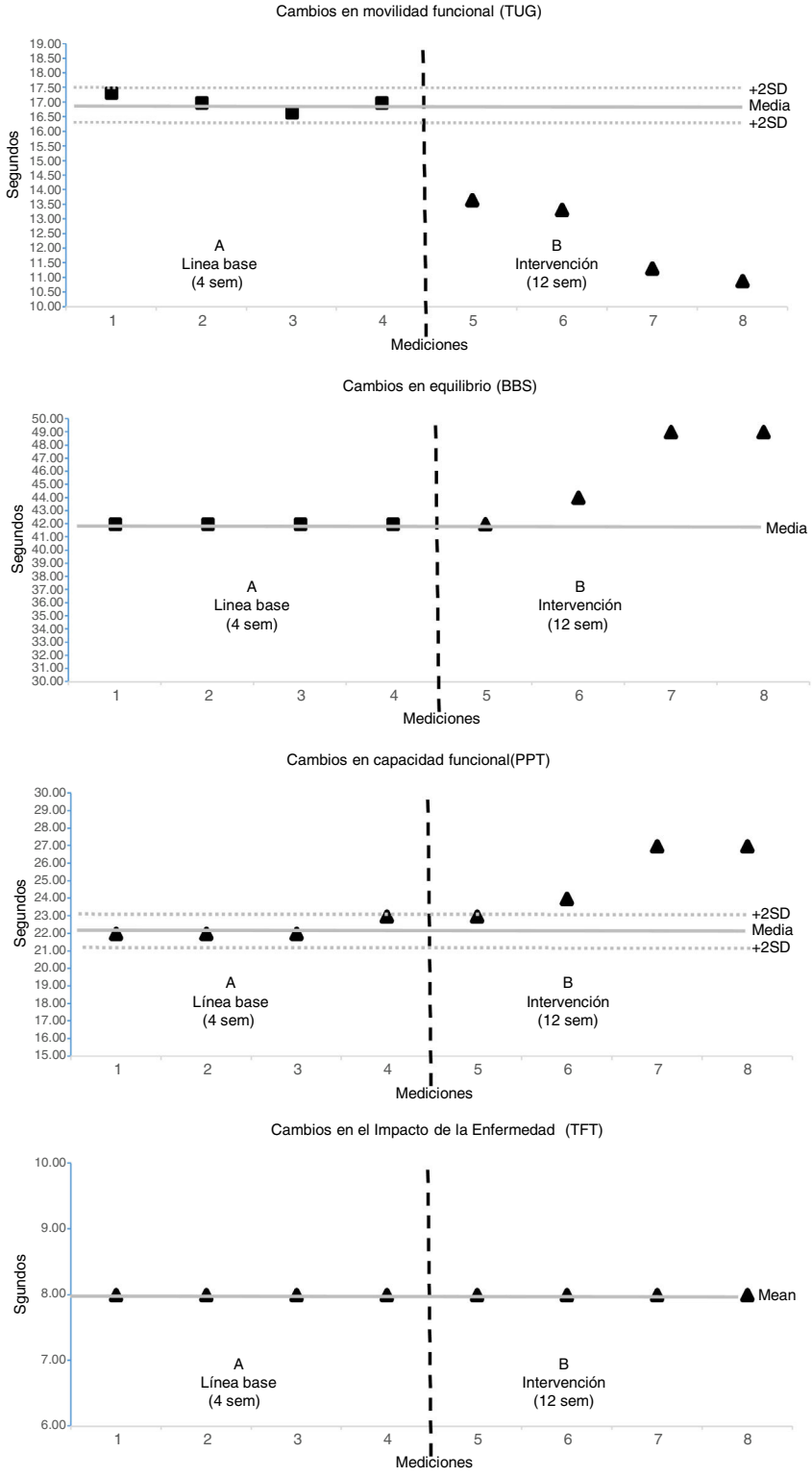

Figure 1 Visual analysis of 2 standard deviations to assess changes in functional mobility, balance, functional capacity, and impact of the disease before and after the intervention.

Table 1 Comparison of functional status and independence before and after the intervention.

	MA ± SD (min-max)	MB ± SD (min-max)	Improvement (%)	Trend	Overlap (n)	Change
Functional mobility (TUG)	17.0 ± 0.3 (16.7-17.3)	12.3 ± 1.4 (10.9-13.7)	27.6	+	4	4.7
Balance (BBS)	42.0 ± 0.0 (42.0-42.0)	46.0 ± 3.5 (42.0-49.0)	9.5	+	3	4.0
Functional capacity (PPT)	22.2 ± 0.5 (22.0-23.0)	25.2 ± 2.1 (23.0-27.0)	13.5	+	3	3.0
Impact of the disease (TFCS)	8.0 ± 0.0 (8.0-8.0)	8.0 ± 0.0 (8.0-8.0)	0.0	—	0	0.0

BBS: Balance Berg Scale; MA: mean in phase A; MB: mean in phase B; PPT: Physical Performance Test; SD: standard deviation; TFCS: Total Functional Capacity Scale; TUG: Timed Up and Go Test.
To determine the effect of the intervention, we performed a visual analysis of single-case data during the 2 phases: initial assessment (A) and intervention (B); to do this, we analysed improvement, trend, overlap, and change between phases. Improvement: result of the comparison between means for the A and B phases. Trend: (+) positive differences between A and B phases; (—) no differences between A and B phases. Overlap: total number of assessments during the B phase that met 2 conditions: *1)* different from and better than A, *2)* the 3 latter measurements suggest improvements. Change: differences between the mean for the A phase and the last assessment (week 12).

University of Vigo for information on the types of physical therapy she could receive, the patient agreed to participate in a 12-week Nordic walking programme (3 sessions per week). We conducted a repeated measures single-case study following an A-B design.[4] During the A phase, the patient was assessed once per week for 4 weeks. During the B phase, the patient completed the Nordic walking programme, with assessments performed weekly. The School's Ethics Committee approved the study, and the patient gave informed consent to participate. In the first sessions the patient was taught the Nordic walking technique and took 12-minute walks, which were gradually increased to 25 minutes' duration. The Timed Up and Go Test (TUG), Berg Balance Scale (BBS), and Physical Performance Test (PPT) were used to assess the effects of the programme on functional mobility, balance, and physical functioning, respectively.[5] To identify possible changes in disease progression, we used the Total Functional Capacity Scale.[6] The patient completed all the scheduled sessions (n = 36) and presented no adverse effects. The distance covered per session increased from 700 to 1830 m, and mean speed increased from 3.9 to 4.5 km/h. Assessment results suggested an overall improvement in all variables, with the exception of disease progression (Fig. 1). The total time needed to complete the TUG decreased to 4.6 seconds (an improvement of 27.6%). BBS and PPT scores increased by 4 and 3 points, respectively, which represented an improvement of 4% and 3% (Table 1). Our results are consistent with those of other authors reporting the effects of physical exercise on HD. For instance, Mirek et al.[7] applied a programme based on balance and gait, and Piira et al.[8] developed a multidisciplinary rehabilitation programme; both groups detected significant changes in participants' TUG and BBS scores. Similarly, Ciancarelli et al.[9] report improvements in patients' functional capacity (PPT scores) after participating in a neurorehabilitation programme. Despite the potential benefits observed after the intervention, we should underscore 2 points. Firstly, the programme had no effect on disease progression. Secondly, only the reduction in the time needed to complete the TUG can be considered a clinically significant change derived from the Nordic walking programme.[10] In view of these results, we conclude that Nordic walking represents an effective physical rehabilitation strategy for patients with early-intermediate stage HD. Its practice may lead to clinically relevant changes in functional mobility, as well as some improvements in balance and functional capacity. However, these changes have no impact on disease progression.

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Posterior circulation ischaemic stroke as a complication of bronchial artery embolisation☆

Ictus isquémico en territorio posterior como complicación de embolización de arterias bronquiales

Dear Editor:

Bronchial artery embolisation (BAE) is considered the most effective and least invasive technique for the treatment of massive or recurrent haemoptysis. Surgery is the treatment of choice only in cases of chest trauma, iatrogenic pulmonary artery rupture,[1] or complications from previous BAEs. The most frequent complications of BAE are transient chest pain and dysphagia, reported in 1.4%-34.5% and 0.7%-30% of cases, respectively. Other complications include postembolisation syndrome (leukocytosis, fever, pain), vascular injury (vasospasm, dissection, perforation), haematoma, and pseudoaneurysm at the puncture site.[2]

The most frequent neurological complication is spinal cord ischaemia secondary to embolisation of the anterior spinal artery, with involvement of the artery of Adamkiewicz in up to 6.5% of cases.[2,3] A rare, puzzling complication of BAE is ischaemic stroke, most frequently affecting the vertebrobasilar territory. In a literature search, we identified 9 reported cases.[4–11]

We present the case of a patient treated with BAE who presented an ischaemic stroke in the territory of the posterior circulation, and review the possible pathogenic mechanisms of this phenomenon.

Our patient was a 57-year-old woman, a former smoker, with history of type 1 diabetes mellitus, ankylosing spondylitis, and bronchiectasis associated with recurrent haemoptysis. She had undergone BAE 3 years previously without complication. Another BAE was scheduled due to recurrence of haemoptysis. An angiography study of the bronchial arteries was carried out in order to perform selective microcatheter embolisation with 400-μm polyvinyl alcohol particles. During the procedure, the patient presented headache, nausea, vomiting, and decreased visual acuity, associated with high blood pressure (180/90 mmHg) and hyperglycaemia (400 mg/dL). After the procedure, she displayed dysarthria and ataxic gait. An emergency head CT scan revealed hypodense lesions in both cerebellar hemispheres, with no contrast uptake. A CT angiography study of the supra-aortic trunks and circle of Willis revealed no significant pathological findings or anatomical variants at the origin of the vertebral arteries. A brain MRI scan showed multiple acute ischaemic infarcts in both cerebellar hemispheres, the right middle cerebellar peduncle, and extensive areas of the splenium of the corpus callosum (Fig. 1). The remaining complementary tests (complete blood count, biochemistry study, coagulation study, electrocardiography, transthoracic echocardiography, and 24-h Holter monitoring) showed no significant alterations except for known hyperglycaemia and glycosuria. Pulmonary angiography found no vascular shunts.

Symptoms improved during hospitalisation, but unsteady gait and subjective visual alterations persisted at discharge.

The patient has subsequently presented further episodes of mild haemoptysis, which resolved with medical treatment. Although the embolic source of stroke could not be identified, the pulmonology department contraindicated any further BAE due to the episode described previously, although the available literature has not established any formal contraindications.

Cerebrovascular embolism is a rare complication of BAE; some review articles report a frequency of 0.6%-2%.[2]

Most of the cases reported to date have been described in patients with chronic pulmonary disease, which is associated with the formation of shunts between the pulmonary and the systemic circulation; most of these cases present involvement of the posterior cerebral circulation. Several hypotheses may explain the predisposition for this territory.

The following pathogenic mechanisms have been proposed to explain intracranial embolisation in patients undergoing BAE:

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