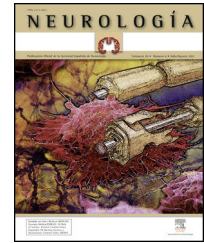


NEUROLOGÍA

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LETTERS TO THE EDITOR

Neuropsychiatric symptoms as a manifestation of multiple sclerosis after 2-year follow-up period[☆]

Sintomatología neuropsiquiátrica como única manifestación de esclerosis múltiple tras 2 años de seguimiento

Dear Editor:

Multiple sclerosis (MS) is a chronic inflammatory disease of the CNS. Its most frequent and typical initial forms of presentation are neuritis optica or arrays of sensory-motor symptoms. Nevertheless, a small minority of patients (about 1%) experience changes in mood, behaviour, personality, or cognition as initial manifestations of MS.¹ This may result in an erroneous diagnosis of a primarily psychiatric condition.

In contrast, as cases of MS progress, neuropsychiatric symptoms are very likely to appear; estimates indicate that they will affect two-thirds of all patients at some point. Depression is by far the most frequent symptom, and occurs independently of the degree of neurological disability.^{3–6} Psychotic symptoms have the same incidence in the general population as in patients with MS.⁵ Psychiatric comorbidities in MS are probably underdiagnosed, and therefore also undertreated.^{3,4} A depressive syndrome is frequently interpreted as a secondary effect of certain drugs or mistaken for MS-associated fatigue.⁴

It should be noted that psychiatric symptoms are not included in Kurtzke's Expanded Disability Status Scale (EDSS), and that cognitive function is represented in a very limited way (as 1 of 8 functional systems). However, both factors have a large impact on the quality of life of MS patients and their families, and they are also associated with an increased risk of suicide.^{2,3,6,7}

We present here the clinical case of a patient who was eventually diagnosed with MS after an onset characterised by neuropsychiatric symptoms. After 2 years of follow-up, the patient continues to have exclusively psychiatric and cognitive symptoms.

Our patient was a 47-year-old female, previously healthy and independent, with no relevant personal or family history.

She was admitted to the psychiatric unit of our local hospital in February 2010 with manic symptoms that had been developing over several weeks. The patient presented euphoria, unexplained laughter, emotional lability, sexual disinhibition, uncharacteristic drug abuse, tachypsychia, verborrhea, racing thoughts, work-related paranoia, and spiritual and hyper-religious ideation (she amassed a large collection of pamphlets issued by religious sects and donated sizeable sums of money to those sects).

Doctors consulted the neurology department due to abnormal findings in the routine CT scan taken when the patient was admitted. CT showed patchy hypodense areas in the white matter in both hemispheres, as well as a degree of cerebral atrophy that was unusual given the patient's age.

Cognitive assessment could not be performed during our initial clinical examination because the patient was experiencing verborrhea and akathisia, with racing thoughts that always led to spiritual topics. Otherwise, the neurological examination showed no alterations to cranial nerves, strength, deep tendon reflexes, plantar reflex, or the 3 types of sensation. No sensory level and no limb dysmetria were present. The patient claimed not to have experienced sphincter dysfunction or any past episodes that might suggest demyelinating episodes. T2-weighted images from a contrast-enhanced brain MRI taken in February 2010 showed numerous hyperintense lesions typical of multiple sclerosis in both cerebral hemispheres, the semioval centre and corona radiata, periventricular area, and the temporal lobes and corpus callosum. Oligoclonal bands were detected in cerebrospinal fluid. A second cerebral and spinal MR scan was taken 6 months later. Images revealed new lesions in both cerebellar hemispheres, the right hemi-pons, and the right anterolateral quadrant of the medulla. Autoimmune and serology studies consistently delivered normal results.

After the patient was discharged in March 2010, her clinical progress was marked by strictly normal neurological findings coupled with abnormal psychiatric and cognitive findings. She suffered a relapse with anxiety as the main symptom and was admitted to the psychiatric unit a month later. Furthermore, ever since she was discharged, she has cited poor performance of daily activities, indicating that she needs her daughter's supervision to complete tasks. This symptom lessened somewhat when neuroleptics were discontinued and replaced with pregabalin and lormetazepam. The patient lives alone at present. She is able to complete most domestic tasks independently, although her pace is

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slowed by her difficulty organising her thoughts. She has had to delegate all administrative tasks to her daughter, needs to carry a planner at all times, and is unable to return to her job (jewellery shop assistant) because of difficulties counting money and following clients' conversations.

Her first valid neuropsychological examination, completed once psychiatric symptoms had stabilised, yielded a score of 28/30 on the Mini Mental State Exam (MMSE). Language and gestural/constructional praxis were spared. Specific neuropsychological test batteries evaluating executive functions, memory, and attention/concentration detected moderate impairment.

In this case, insidious neuropsychiatric symptoms developed over a 2-year course. Unlike in cases in which functional systems included in the EDSS are impaired, determining to what extent the disease may have progressed is difficult here. Although the patient's manic symptoms were florid and even required a hospital stay, we do not regard this event as a first attack. Contrast-enhanced cerebral MR in the acute phase ruled out inflammatory activity.

There is radiological evidence of dissemination in time and space: the first scan showed 3 out of the 4 Barkhof criteria, and the 6-month assessment showed 4 out of 4. In progressive forms of MS like the one described here, neurological damage tends to persist without there being any obvious inflammatory activity. Laboratory tests (oligoclonal bands in cerebrospinal fluid) also supported the diagnosis of MS. Additional complementary examinations were able to rule out other possible diagnoses. Differential diagnosis would not be completed without considering neurolupus, which often manifests with psychiatric symptoms. It was ruled out based on the patient's consistently normal autoimmune parameters and lack of systemic signs and symptoms.

The patient met the McDonald MRI criteria, and was therefore diagnosed with primary progressive MS. At present, her score on the EDSS is 3.0, reflecting moderate disability for one functional system, namely cognition.

Euphoric/manic episodes in MS have been shown to be associated with severe and progressive forms of the disease, associated cognitive impairment, frontal demyelination, cerebral atrophy, and a prolonged course of the disease. The latter description does not apply to our patient. Although

euphoria and other psychiatric disorders are unusual as initial manifestations of MS, we feel that this diagnosis should be considered during initial evaluations of patients in psychiatric units.¹

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¹ Dr L. Fratalia currently works for NHS in England through a private agency. However, follow-up on this patient and all use of resources connected with the case took place exclusively at Hospital de Manacor, Spain.

Intravenous thrombolysis in capsular warning syndrome: is it beneficial?[☆]

Trombólisis intravenosa en el síndrome de alarma capsular: ¿es beneficiosa?

Capsular warning syndrome (CWS) is characterised by repeated and self-limiting episodes of motor deficit (sensory deficit is less frequent), which are caused by ischaemia

limited to the internal capsule. The risk of infarction following CWS is higher than in the case of other transient ischaemic attacks (TIA).¹ CWS treatment during the acute phase is controversial. The effectiveness and safety of intravenous thrombolysis (IVT) is not well-known.^{2,3}

We present the case of a man aged 52 with a history of smoking, alcohol abuse, and obstructive sleep apnoea syndrome (OSAS), who was undergoing treatment with CPAP. He experienced 2 sudden-onset transient episodes of left-sided weakness and difficulty articulating words, each lasting less than 30 minutes. He was treated in his referral hospital, where he suffered a third identical episode before being sent to our hospital. On the way, he suffered another episode in the ambulance. Upon arrival at the hospital, he presented mild right faciobrachiorural hemi-

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