The literature on the subject suggests an association between the neurotoxicity generated by beta-lactam antibiotics and their ability to interact with gamma-aminobutyric acid (GABA) receptors.[6] However recent studies in rats suggest the involvement of other factors, such as increased release of excitatory aminoacids,[7] which has yet to be demonstrated.

Considering the above, we conclude that in patients who are receiving treatment with carbapenem and present neurological alterations or altered mental state, especially in cases of patients with CKD or elderly patients, we should consider an adverse effect of the drug to be a potential cause after ruling out other possibilities. According to the pattern observed in these cases, symptoms can persist up to 2 weeks after suspension of the drug.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Logopenic progressive aphasia associated with idiopathic Parkinson's disease☆,☆☆

Afasia progresiva logopénica asociada a enfermedad de Parkinson idiopática

Dear Editor,

Primary progressive aphasia is a clinical syndrome characterised by a language deficit of neurodegenerative origin with no other cognitive manifestations, at least during the initial stages.[1] Three clinical variants have been described to date (nonfluent, semantic, and logopenic), each associated with its distinct topography and anatomical pathology.[3] Of the 3 variants, logopenic aphasia is mainly associated with Alzheimer disease, and it is considered an atypical form of AD onset.[1] However, the association between logopenic aphasia and Alzheimer disease is still a matter of debate in the literature. Associations with other diseases have been found in a high percentage of cases in studies using molecular imaging, cerebrospinal fluid biomarkers, or anatomical pathology findings.[3,4] Furthermore, cognitive impairment and dementia associated with Parkinson's disease are regarded as frequent. They are characterised by executive and/or memory deficits, while language typically remains preserved.[5] We present the case of a patient with idiopathic Parkinson's disease who developed symptoms of progressive logopenic aphasia. Her symptoms finally progressed to generalised dementia with biomarkers of Alzheimer disease, thereby supporting the idea that this type of aphasia is a marker of Alzheimer disease.

Our patient is a 65-year-old woman with high blood pressure and dyslipidaemia, who in 2009 was diagnosed with idiopathic Parkinson's disease after a one-year period in which she displayed slowness and tremor. Neurological examination revealed rigidity, bradykinesia, and resting tremor predominantly on the right side. She responded favourably to levodopa and subsequently developed motor fluctuations. She also presented REM sleep behaviour disorder. Since mid 2012, the patient began reporting increasing difficulty finding words without any associated memory impairment or behaviour disorder. She experienced no hallucinations and her neurological examination did not

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☆☆ This study was presented at the 65th Annual Meeting of the Spanish Society of Neurology (Barcelona, 19—23 November 2013).

reveal oculomotor alterations, pyramidal signs, or cerebellar signs. Neuropsychological evaluation performed in early 2013 showed qualitative impairment of language fluency consisting of frequent pauses to find words, with no aphasic transformations and well-articulated speech. We observed anomic aphasia both in visual-verbal naming and verbal-verbal naming, and conduction aphasia, with preserved verbal comprehension, grammatical structures, and semantic usage. She also presented a mild divided attention deficit (TMT-Part B). Verbal memory tested as borderline, while the rest of the evaluation yielded normal results (Table 1). The PET-TC study showed left temporoparietal hypometabolism (Fig. 1) and magnetic resonance scan of that region revealed asymmetry. Analysis of cerebrospinal fluid showed a decreased concentration of A-β42 proteins (199 pg/mL, normal value > 500 pg/mL), increased T-protein (443 pg/mL, normal value < 400 pg/mL), and increased phosphotau levels (62 pg/mL, normal value < 61 pg/mL). Over the following months, the patient's clinical symptoms progressed and her speech symptoms grew more marked. Memory deficit, disorientation, and functional decline also began to manifest.

The main signs of aphasia and findings from the FDG-PET study met diagnostic criteria for the logopenic variant of primary progressive aphasia,[2] which subsequently progressed to overall cognitive impairment. In this case, we

Table 1 Results from the neuropsychological evaluation.

	Raw score	Scaled score[b]
MMSE	26/30	
Addenbrooke's Cognitive Examination	63/100	
Orientation	35/35	18
Boston Naming Test	31/60	5
Verbal-verbal naming. Answer the question[a]	11/18	Deficient
Verbal-verbal naming. Complete the sentence[a]	18/18	Normal
Token Test	30.5	8
Word comprehension[a]	36/36	Normal
Word comprehension (body parts)[a]	18/18	Normal
Comprehension of instructions[a]	14/16	Normal
Repetition of syllables[a]	8/8	Normal
Repetition of syllable pairs[a]	8/8	Normal
Repetition of logotomes[a]	8/8	Normal
Word repetition[a]	10/10	Normal
Repetition of sentences[a]	44/60	Deficient
Pyramids and palm trees test	47/52 (90.4%)	Normal
Orobuccal apraxia[a]	20/20	Normal
Direct and inverse verbal span	5/3	10/9
Trail Making Test A/B	106/356	5/4
Stroop A/B/C	63/31/26	7/5/9
Symbol Digit Modalities Test	19	8
Symbolic gesture, on command and imitation (one hand)[a]	10/10	Normal
Mimicry of object use, on command and imitation[a]	10/10	Normal
Imitation of postures: unilateral, bilateral[a]	10/10; 7/8	Normal
Basic constructive praxis: copying[a]	14/18	Normal
Rey-Osterrieth complex figure: copying	30	9
Rey-Osterrieth complex figure: 3 min	6.5	7
Rey-Osterrieth complex figure: 30 min	6.5	7
Free and Cued Selective Reminding Test. Total free recall	10	6
Free and Cued Selective Reminding Test. Total recall	28	6
Free and Cued Selective Reminding Test. Delayed free recall	4	6
Free and Cued Selective Reminding Test. Delayed total recall	8	6
Semantic fluency task (animals in 1 min)	8	3
Lexical fluency task (words beginning with 'P' in 1 min)	7	7
Tower of London test. Correct movements	2	8
Visual object and space perception battery. Object decision	17	11
Visual object and space perception battery. Progressive silhouettes	12	10
Visual object and space perception battery. Position discrimination	18	6
Visual object and space perception battery. Number location	10	18

[a] Subtest from the revised Barcelona Test.
[b] According to patient's age and years of education (8 years), using normative data from Neuronorma project. A scaled score less than or equal to 5 is considered deficient.[10]