Does cognitive profile distinguish Lewy body disease from Alzheimer’s disease in the early stages?

F Blanc

Lewy body disease (LBD) is the second most frequent neurodegenerative disease after Alzheimer’s disease (AD). To distinguish these two pathologies seems to be of less importance at this time since AD and LBD have the same symptomatic treatment (cholinesterase inhibitors). However, new specific treatments emerge in AD with precise targets against brain lesions. Moreover frequent symptoms of LBD such as hallucinations and delusions do not have to be treated by usual neuroleptics and antipsychotics, since such treatment aggravate patients physically and cognitively.

The diagnosis of LBD is difficult for clinicians, even for specialists, particularly at the beginning of the disease. The diagnosis criteria, known as McKeith criteria, have a really high specificity—more than 95%—for LBD dementia. The sensitivity of such criteria was found to be 83%, using a cohort of 50 patients. In a bigger cohort of 2861 patients, with less strong clinical data but probably better reflecting the real world of clinical diagnosis, the sensitivity of the diagnosis of LBD was 32% (and when LBD is mixed with AD, it was 12%). These last results mean that the majority of the LBD patients—with dementia and mild cognitive impairment (MCI)—are not diagnosed. These poor results can be explained by the fact that brain MRI shows unspecific atrophy and usually any atrophy at the beginning of the disease. In the same way, brain perfusion SPECT can show occipital hypoperfusion but only for 65% of the patients, and for patient with moderate dementia (patients with a mean Mini-Mental State Examination of 16/30, and with a mean history of DLB of 5 years). And finally, (123)FP-CIT SPECT is helpful in differentiating dementia with LBD from AD, but not with LBD from frontotemporal lobe dementia. This last SPECT is also probably little used because of the cost. That is the reason why we really need more clinical data to better diagnose LBD patients, particularly at the beginning of the disease.

In the paper by H Yoshikawa et al., DLB patients (n=12) were compared to AD (n=89), and mixed AD and DLB (n=23) patients at the early stage of disease (MCI or mild dementia with clinical dementia rating (CDR) of 0.5 or 1—only one patient with AD had no cognitive impairment with CDR=0). The strength of the study is that all cases have a neuropsychological diagnosis. First, the authors found that at the early stage of the disease, DLB patients have more visuospatial deficits than AD and AD+DLB patients, using the Rosen drawing test (RDT). In this test, subjects copy five visual designs, and they obtain one point for each design correctly copied. This test is not only a visuospatial test but a constructional test, including also strategy and planning functions such as the copy of the Rey-Osterrieth complex figure. In the only previous study with clinico-pathological data on prospectively followed patients with MCI and LBD, visuospatial functioning was impaired in six out of eight patients, at least for one of the three different cognitive tests used (Block Design and Completion Subtests of the Wechsler Adult Intelligence Scale-Revised and copy of Rey-Osterrieth complex figure). Moreover, patients with MCI and AD were found by Yoshikawa et al., to have more memory storage impairment, using the delayed recognition score of the SRT. Such memory impairment is well known in prodromal AD, as previously described by Sarasin et al. Using these two cognitive results—that is, the RDT and the delayed recognition of the SRT, Yoshikawa et al. have shown that the prediction of DLB compared with AD (sensitivity 83%, specificity 85%) and the prediction of DLB compared with DLB+AD (sensitivity 92%, specificity 83%) were good, but not the discrimination of DLB+AD from AD (sensitivity 61%, specificity 57%).

Such cognitive profile was previously described for DLB dementia and is now demonstrated for DLB MCI thanks to this autopsy confirmed study.

To diagnose LBD at the early stage is still a challenge, and clinicians have to search actively also for cardinal signs of LBD (fluctuations, discrete signs of parkinsonism, visual hallucinations and illusions). Other symptoms seem also to argue for the diagnosis of LBD at the early stage such as constipation and increased saliva.

Competing interests FB has received honoraria as member of board from Eisai, as expert from Biogen, Roche and Piramal, for lecture from Janssen, Eisai, Pfizer, Novartis, Lundbeck and Biogen.

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