IS MILD COGNITIVE IMPAIRMENT A USEFUL CONCEPT – YES

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The concept of Mild Cognitive impairment (MCI) evolved from epidemiological studies in elderly populations showing a large number of persons with cognitive impairment without dementia, and from clinical observations that memory and other cognitive complaints often precede dementia. There are large variations in the rate of progression towards dementia in persons with MCI, depending on where they come from: highest in specialized memory clinics, lowest in random population samples. MCI has even been found to be reversible for some subjects in population-based studies.

MCI is considered as a risk state for dementia, particularly Alzheimer's type (AD). This risk is higher in persons carrying the ApoE4 genotype, but this is no justification for routine genotyping in clinical practice. Clinicians can use the Mini Mental State Examination (usually normal in MCI) supplemented by the Montreal Cognitive Assessment or similar office screening test; if abnormal patients can be referred for further neuropsychological assessment exploring various cognitive domains. MCI subjects are then classified as 'predominantly amnestic' vs 'non-amnestic', 'single domain' vs 'multiple domain', although it is not yet clear which profile best predict progression to AD. Behavioral symptoms (depression, anxiety) are also common in MCI and may also be a forewarning of dementia. The lack of significant impact of the cognitive symptoms on day to day functioning is the current distinction between MCI and dementia; clinicians must explore activities of daily living with the help of an informant, including complex hobbies and decision-making abilities at work and at home.

In terms of treatment, cholinesterase inhibitors have not been found conclusively to alleviate the mild cognitive symptoms nor to delay progression to dementia. There is an ongoing effort to distinguish among persons with MCI who has 'pre-dementia AD', using clinical criteria (amnestic syndrome) supported by neuroimaging (MRI showing hippocampal atrophy; PET with FDG showing parieto-occipital hypometabolism; PET with PIB showing amyloid accumulation) and/or biochemical changes (CSF showing low aβ42 and high tau levels), in order to initiate randomized clinical trials using AD-modifying drugs. Until such 'secondary prevention' studies prove effective, subjects with MCI should be followed longitudinally for being at risk of dementia, treating coexisting conditions such as depression, metabolic or endocrine deficiencies, upper airway obstruction, avoiding drugs with anticholinergic side-effects, and treating vascular risk factors for dementia (diabetes, hypercholesterolemia, systolic hypertension).

There is thus no doubt that MCI is a useful concept, both for clinical practice and clinical research: it may offer the best hope for secondary prevention of AD.

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