QUESTION-AND-ANSWER SESSION

Q: Studies are showing that treatment initiated when pre-dementia or early memory loss is recognized leads to a better prognosis than waiting to initiate treatment once Alzheimer's disease (AD) or dementia is established. What options do clinicians have to address early memory loss?

Drs. McCaddon and Hudson: Clinical options for early memory loss comprise three broad categories: treating vascular risk factors, providing neuroprotection, and promoting neuronal reserves.[1] Vascular risk factors include hypertension, hypercholesterolemia, diabetes, smoking, and hyperhomocysteinemia, all of which should be actively screened for and addressed in all patients presenting with cognitive impairment. Neuroprotection includes ensuring adequate B_{12} and folate status, use of antioxidants, and addressing neuroinflammation. There is also evidence that neuronal reserves can be developed by encouraging cognitive, physical, and social activity.[1]

Q: Are there symptoms or markers that can be used to diagnose pre-dementia or early memory loss?

Drs. McCaddon and Hudson: There is a pathway of cognitive impairment where benign aging develops into mild cognitive impairment (MCI) and eventually AD. Early recognition of this trajectory is vital if the process is to be slowed or halted. Mitrushina and colleagues[2] reviewed neuropsychological tests used for the assessment of MCI and AD.[2] Many of these are time-consuming to perform and impractical in the busy physician's office. In addition, fatigue and sensory loss may impair the ability of the very elderly to complete these tests.[3] Milne and colleagues[4] reviewed shorter tests more suitable for use by the primary care physician, such as The General Practitioner Assessment of Cognition, the Memory Impairment Screen, and the Mini-Cognitive Assessment Instrument, and there is good agreement between these and the Mini-Mental State Examination.

Despite the wide variety of cognitive function tests available to the physician, anatomical and metabolic changes in the brain, demonstrated by magnetic resonance imaging and positron emission tomography, already occur ≥2 more years before a diagnosis of MCI can be made.[5,6] Biomarkers in cerebrospinal fluid, such as tau protein and amyloid-β, are promising candidates as early markers but await long-term studies of their potential clinical utility.[7] In summary, there are several techniques available to the physician for the detection of symptomatic MCI or AD. The detection of pre-clinical disease remains a challenge, but is a focus of much current research.

Q: What is your goal when treating patients with pre-dementia or early memory loss?

Drs. McCaddon and Hudson: The main goal is to delay or possibly even halt cognitive decline. Anything that might delay or prevent the onset of overt dementia would be beneficial from both an individual and epidemiological viewpoint. Remarkably, it is estimated that dementia prevalence would be halved if risk reduction strategies delayed the onset of dementia by only 5 years.[8]

Q: How important is safety and tolerability in a therapy used to address early memory loss?

Drs. McCaddon and Hudson: Safety and tolerability are extremely important in all patients, perhaps more so in those presenting with early memory loss. Cerefolin NAC is well tolerated and not associated with any significant drug interactions. It is also easy and convenient to use, being a single dose caplet with no necessary dose adjustment. In addition, in a recent cross-sectional study the relationship between folate and risk of cognitive impairment is reversed in patients with low B_{12}; high folate, and low B_{12} concentrations are associated with an increased risk of cognitive impairment.[9] Although this association requires further investigation, Cerefolin NAC ensures that patients receive an optimal balance of the two vitamins.

Q: In your opinion, where does a novel therapy like Cerefolin NAC fit in the options available for memory loss?

Drs. McCaddon and Hudson: Cerefolin NAC is a useful option for early memory loss because it offers a synergistic approach to neuroprotection. A recent study confirmed that many patients with dementia have brain changes consistent with both AD and vascular dementia.[10] The authors suggested that it may be necessary to develop combination therapies to treat dementia. A similar synergistic approach should perhaps also be considered in patients presenting with early memory loss.

Reference List

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https://doi.org/10.1017/S1092852900027590 Published online by Cambridge University Press