Sensory impairments and cognitive disorders in older age

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Abstract

Age-related and neuropathological changes in the olfactory, visual, auditory, and motor systems suggesting that sensory and motor changes may precede the cognitive symptoms of Alzheimer’s disease (AD) by several years and may signify increase the risk of developing AD. In particular, peripheral age-related hearing impairment and social isolation have been identified as potentially modifiable dementia risk factors. The impact of age-related hearing and vision impairments on cognition appeared to be especially important among the oldest individuals. It is possible that the association is not specific and not unique to one sensory system suggesting therefore that the presence of functioning sensory systems may be a marker of healthy brain aging as suggested recently by the investigators of the epidemiology of hearing loss study (EHLS), a longitudinal, population-based study of aging in the Beaver Dam, a community in Wisconsin. In this study, hearing and vision impairments were associated with a relative risk around two with cognitive impairment while olfactory loss with a relative risk of four (5). In this study, however, 85% of participants with hearing impairment, 81% with visual impairment, and 76% with olfactory impairment did not develop cognitive impairment during follow-up. This selected risk for a segment of the population clearly indicated the presence of some other factors that may modulate the risk of cognitive impairment in presence of specific sensory deficits. A recent meta-analysis of studies on retinal thickness including 887 AD patients, 216 patients with mild cognitive impairment (MCI), and 864 healthy controls suggested a clear reduction of total macular thickness in patients with AD. Another layer of interest could be represented by new biomarkers for both early diagnosis and pathogenesis of disease. In the last few years, several ocular clinical symptoms and signs in AD have been reported: β-amyloid (Aβ) accumulation in the retina and in the lens, fiber layer loss in the optic nerve, and especially, a wide range of retinal vascular pathological changes. Many of these changes have been proposed as potential biomarkers of AD. One of the most interesting vascular markers is retinal blood flow. In a recent study, blood column diameter, blood speed, and blood flow have been measured in a major temporal retinal vein using retinal laser Doppler flowmetry in AD, MCI, and control subjects. In addition, peripapillary retinal nerve fiber layer (RNFL) thickness was measured using optical coherence tomography (OCT). Blood flow had intermediate values in MCI subjects between what was measured in controls and AD patients suggesting the importance of vascular changes in determining neurodegeneration. In patient with clinical AD, the RNFL and macular thickness measured by spectral domain-OCT (SD-OCT) were significantly thinner compared to controls. The macula is thinner as well in all sectors except the fovea compared to controls. Macular measurements may be reliable indicators of visual impairment in AD and vision should be therefore checked in these patients. The most relevant issue is
that visual dysfunction in AD patients can now be considered as direct outcome of retinal abnormalities due to the deposition of specific AD pathology in the retina. Elevated Aβ42/40 peptides, several types of Aβ plaques, and phospho-tau protein can be found in the retina with appropriate neuropathological techniques.9 In line with the above findings, in animal models have described retinal Aβ deposits and tauopathy.9 As in specific areas of the brain the amyloid and tau deposits are associated with local inflammation, retinal ganglion cell degeneration may be an additional finding that could explain visual functional deficits in AD.10

References