

Alzheimer's disease: Retinal Scanning looking towards the future

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The soul can speak through the eyes and so can the mind. If we could just look into the vast array of information that is waiting to be retrieved with only a glance, how many lives could be changed for the better, if we could detect Alzheimer's disease (AD) in the process? The eye could be the 'window' into the brain that will enable early detection of change, while providing valuable insight into existing and future risks for developing AD.

Dementia is the single largest health issue facing Australia in the 21st century, with 50 -70% of all dementia being of the AD type. The economic impact of dementia on the health care system, while currently dire is projected to potentially cripple global economies by 2050. The total estimated worldwide cost of dementia in 2010 was US\$604 billion and by 2050 it is predicted to increase to \$1 trillion per annum. By 2050 within Australia the projected burden will be a staggering \$83 billion, equivalent to approximately 11% of the entire Australian health/residential aged care sector expenditure. Currently more than 266,574 people in Australia have dementia and this is projected to increase to 942,624 by mid-century (Access Economics 2011).

On a social and personal level AD is a progressive neurodegenerative disease, which progressively robs people of their memory, ability to reason, solve problems, communicate and undertake many activities that form our daily lives. This disease is not a normal part of ageing and although it is more common in the aged, it also occurs in younger generations. A MRI or CT scan will clearly demonstrate the brain shrinkage that occurs in a person who has AD, which results from neuronal cell death, but this is a very late indication of disease and treatment at this stage is, too little, too late.

Missing the diagnosis

Clinical experience in the field of dementia diagnosis and management in a range of settings, including acute, rehabilitation, residential and scientific arenas, cements the notion that the process of AD confirmation can be laborious, even for the most experienced clinician. As with all diseases, the clinician generally relies upon a battery of tests to elicit an accurate diagnosis, however, in the case of dementia, the diagnosis is still a process of excluding any other potential conditions. To comprehensively assess for dementia the clinician undertakes a thorough physical and neuropsychological examination, health history, medication review, blood testing to check for deficiencies or imbalances, informant history and brain imaging. Dependent on the resources of the clinician and also often the age of the patient, additional specialised testing may be explored.

Imaging procedures such as the Positron-emission tomography (PET) scan, used in conjunction with a special marker substance, can now measure accumulated beta amyloid plaques. These plaques, which can be described as clustered clumps of protein, are believed to be a pathological feature of AD and considerable effort has been focused on reducing, clearing or slowing the accumulation of this protein in the brain. While this technology exists it is expensive and generally only available through clinical research or similar specific circumstances.

Worldwide there is a quest to find additional measures that can supplement the clinicians 'toolbox', so that the diagnosis can be established in a more concise, non-invasive and cost efficient manner.

Why retinal scanning?

While most AD pathology occurs in the brain, changes to the retina early in the disease suggest that Alzheimer's pathology might also occur in the retina, making the eye a window for early detection of the disease. Curcumin, from the Indian spice turmeric, has recently been investigated for its ability to bind or stick to beta-amyloid plaques. Oral formulas containing curcumin have been optimised to pass the blood brain barrier, bind to plaques and contribute to clearance of plaques. Curcumin is a powerful antioxidant and anti-inflammatory and in addition to its propensity to bind to plaques, it has optical fluorescence properties. This means that under specialised equipment it 'lights up' the plaques in the retina, making it a potential imaging marker for AD.

Previous studies have associated AD with thinning of retinal vessel blood column diameter; thinning of the retinal nerve fibre layer; and loss of retinal ganglion cells. Our previous work has identified many retinal vascular changes in AD, including thinning vessels and changes to the branching geometry of the retinal vessel network. The cause of these retinal vascular changes in AD has so far remained unclear. Significant similarities exist between the retinal and cerebral micro vasculatures, and hence the retinal abnormalities may be related to cerebral microvascular changes in AD. Studies to date, have found evidence of retinal beta-amyloid plaques in animal models of AD and also in the post-mortem human AD retina. This study is also investigating the possibility that beta-amyloid plaques, the hallmark of AD in the brain, are also occurring in the retina and leading to retinal degeneration.

While studies using Curcumin for its therapeutic potential in AD are currently being trialled in two sites, including Sydney and Western Australia, this is the first study of its kind to include retinal scanning as an outcome measure.

Those less informed might suggest that they would rather not know if they had a risk of AD, yet these changes may be the 'warning signs' needed to engender a lifestyle change, that could alter the disease trajectory. One of these changes might be associated with antioxidant intake. According to a report by Access Economics, if the onset of dementia could be delayed by five years the overall incidence could potentially be halved.

Summary

If a consistent relationship can be demonstrated between retinal plaques and those occurring in the brain, utilising Curcumin fluorescence imaging, this could potentially provide a non-invasive population-screening test for AD. In the future, when you go to the ophthalmologist for an eye examination, apart from screening you for macular degeneration, glaucoma, and related diseases, you may even be able to have your risk for AD measured.

Bibliography

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