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Blocking neuroinflammation due to complement activation by amyloid with vaccinia virus complement control protein (vcp), a better alternative to aduhelm in Alzheimer 's disease (ad) due to apoe4 predisposition

Girish J. Kotwal
Kotwal Bioconsulting, USA

Statement of the Problem: Alzheimer's disease (AD) is primarily a progressive neurodegenerative disease accompanied with memory loss that is primarily due to aging although recently AD is considered to be due to genetic predisposition resulting from the presence of ApoE4 allele. AD is caused by abnormal accumulation of beta amyloid and tau proteins in and around neurons in the brain, affecting synaptic junctions. Both beta amyloid and tau activate complement pathways which then results in neuro inflammation. AD related dementia affects 30+ million people globally and the annual cost of care is estimated at half a trillion.

Methodology & Theoretical Orientation: DNA was isolated from saliva of 2 individuals. The sequence of ApoE allele was determined. VCP was purified from overexpression in the yeast system. It was delivered to the brain by 2 routes and found to be effective in transgenic mice in achieving memory protection.. A human trial with a select cohort of about 40 subjects (20 male and 20 female) with homozygous ApoE4 allele above age 55 would be selected. 20 of the subjects will receive VCP and 20 will receive placebo. Various quantitative cognitive and memory parameters will be followed and the benefit of VCP in memory retention will be assessed.

Conclusion & Significance: In light of the limitations of the FDA approved treatment Aduhelm for Mild Cognitive impairment, new treatments such as VCP which can be delivered across the blood brain barrier without invasive procedure need to be included in human trials to prevent AD symptoms by early intervention in persons with ApoE4 allele.

Biography

Girish J. Kotwal, Ph.D. has been working on the Alzheimer's Disease (AD) related neuroinflammation for over 2 decades. He along with his doctoral student James Daly were the first to demonstrate a cause and effect relation between abeta fibrils that contribute to amyloid plaques and neurodefeneration resulting in memory loss and symptoms of AD. He was the first to propose that complement mediated inflammation can be blocked by vaccinia virus complement control protein (VCP). Recently his group has shown that VCP can have improved outcomes in AD mice by delivering without any invasive procedure to the brain, Novel therapeutics like VCP could be a much better and safer alternative to monoclonal antibodies which could have adverse effects by activating the complement pathway thereby increasing neuroinflammation.