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Limitations of Adulhelm for treatment of mild cognitive impairment of Alzheimer's Disease (AD), opens new avenues for other promising treatment candidates

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Aduhelm/Aducanumab is an amyloid beta-directed monoclonal antibody indicated to treat Alzheimer's disease. Aduhelm is approved under the accelerated approval pathway by US FDA. There are multiple limitations in the use of Aduhelm.It is unclear how effective Aduhelm is when it is transfused intravenously (IV) once a month for one hour each time. 2) The initial cost was estimated to be $56,000/year/patient. Later the cost was halved. No idea how many could qualify for Medicare coverage. Could be a million with early stage memory loss or cognitive deficit. 3) Those with moderate to severe cases will not qualify. 4) There are other FDA approved drugs for early stage Alzheimer's disease (AD). 5) The goal of the Aduhelm is to reduce beta amyloid and tau fibril formation or accumulation requiring monitoring with sophisticated imaging that is not cheap. 8) There is no clear assessment of the adverse effects of pushing a monoclonal antibody passed the delicate aging blood brain barrier. 9) Not sure whether the concerns of 2 members of the FDA panel who resigned have been addressed. 10) It is well known that AD patients need considerable care and caring for a patient receiving Aduhelm will add to the care required. There could have been much better ways to deliver Aduhelm that were not tried before choosing the IV route. Also, there are other candidates like vaccinia virus complement control protein (VCP) that can target the harmful effects of amyloid and even prevent death that Aduhelm has been reported to cause. VCP can block both pathways of complement activation and prevent formation of pro-inflammatory chemotactic factors, C3a and C5a and bring about neuroprotection. Extensive pre-clinical studies have confirmed VCP as an ideal candidate to treat AD in humans.

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.