Are there reliable Biological markers for early Alzheimer's Disease?

Magda Tsolaki, MD, PhD,

Aristotle University of Thessaloniki, Chair of Greek Federation of Alzheimer's Disease

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Abstract

Alzheimer's Disease (AD) is defined by the positivity of biomarkers of both amyloidopathy (A1) and tauopathy (T1) in line with the pathologic definition of the disease. The validity of measures is even more essential in preclinical stages, that is, in conditions where there are no other relevant signs helpful for diagnosing the disease. The quality of the measures should be unquestionable. Three phases of the disease can be distinguished in the continuum: The term of Asymptomatic at Risk for clinical AD (AR-AD) when the evolution to a clinical AD is less likely or still needs to be determined (only one pathophysiological marker considered abnormal). Asymptomatic at risk of AD consists of cognitively normal individuals for whom the biomarker pattern is insufficient to reach the above definition of AD. They can be characterized by the positivity of the pathophysiological biomarker (i.e. either "Asymptomatic A1" or "Asymptomatic T1"). The term of "preclinical AD" when the risk is particularly high (e.g., both Aß and Tau markers beyond pathologic thresholds). A clinical stage ("clinical AD") defined by the occurrence of the clinical phenotype of AD (either typical or atypical) and which encompasses both the prodromal and the dementia stages. Topographical markers: Downstream topographical biomarkers (MRI and FDG-PET) are not suitable for defining preclinical AD. However, they may be useful for the screening of subjects at risk. New neuroimaging and magneto-encephalography (MEG)/electroencephalography (EEG) tools can target preclinical disease evolution through structural, metabolic, or functional measurements. To date, evidence from studies on blood-based biomarkers indicates a limited value for the characterization of preclinical stage of AD. However plasma protein biomarkers can be useful to triage potential trials participants for PET or cerebrospinal fluid measures of Alzheimer's disease (AD) pathology. In a recent study seven only from 34 proteins replicated in their ability to predict in vivo amyloid pathology. These proteins form a biomarker panel that, along with age, could significantly discriminate between individuals with high and low amyloid pathology with an area under the curve of 0.74. The performance of this biomarker panel remained consistent when tested in apolipoprotein E ε4 non-carrier individuals only. If blood biomarkers can be used in clinical trials with asymptomatic to risk of AD patients then we can discuss about a hope to find a medication to stop the progression of asymptomatic to symptomatic AD.

Biography:

Dr. Magda Tsolaki, MD, PhD, was born in Thessaloniki, Greece in 1954 and has spent most of her time there. She has been a Professor of Neurology since 2010, a Neuropsychiatrist since 1983 and she has worked at the Aristotle University of Thessaloniki since 1982, as well as at the 3rd Department of Neurology of Aristotle University of Thessaloniki since 1988. She has been the main author/co-author of 48 Books, has participated with 506 abstracts in Greek Conferences, with 403 abstracts in English International or European Conferences, is the first author/co-author of 285 papers in Greek journals, and in 376 international journals -310 in PubMed with Impact Factor=1134.845, h-index=52, with more than 10.445 citations-. She has been a reviewer for Conferences and Journals (261) and has organized 27 national conferences on the AD and five international ones. She was one of the three or seven advisory members for 18 doctoral theses-completed, has created the Greek Alzheimer Association in 1995 and the Greek Federation of Alzheimer's Disease in 2007 - she is also the Chair of this Federation. In this capacity, she has given 310 lectures throughout Greece, as well as on TV and radio. She has participated in 40 research programmes and 37 clinical trials. In total, she has received 48 awards.

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