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Introduction

Alzheimer disease (AD) is a chronic neurodegeneration disease, which it occurs degeneration of the neurons. 60% to 70% of cases of dementia are caused by AD [1]. e early symptom of AD is short-term memory loss and in chronic, it is characterized by language problems, insidious in onset problems, impairment of activities, mood swings, loss of motivation, hoarding, not managing self-care and behavioral issues. Finally, bodies' functions are lost and leading to death. It is mostly occurred in people over 65 years of age [2]. AD is neuropathologically characterized by amyloid beta plaques surrounded by neurons containing neuro brillary tangles in the brain [3]. Formative event in AD, production of Amyloid beta is the result of cleavage of amyloid precursor protein (APP) which amyloid beta is high in AD [4]. APP plays important role in developmental processes in cell di erentiation and establishment of synapses [5] whereas the function of APP is not clear properly in adult brain [4]. Tau protein is an integral part of microtubules in healthy neurons and support to nutrients, vesicles, mitochondria and chromosomes. e hyperphosphorylation of the tau protein results into formation of neuro brillary tangles which aggregates intracellularly and cause neuronal death [6]. In ammation is clearly seen in pathologically vulnerable region of the AD brain [7]. Immune cells as Microglia, astrocytes and neurons are liable for in ammatory reaction that activate and produce in ammatory mediators to clear cellular debris from the damage area [8]. e purpose of this review article is to

complement system in AD [18]. ere are many possible approaches which can Neurons can generate the in ammatory molecules and can serve as source of complement systems and others. Many cytokines produce by neurons or glia, moreover, cytokines are secreted by variety of immune cells, the level of cytokines uctuate in AD brain. e numbers of interactions are reported between cytokines and the components of the AD [4].

Neuroin amation in AD is served to aggravate AD and is promoted the progression of disease. Plaques and peptides of Amyloid Beta are greatly cytotoxic which trigger chronic in ammation in the brain cells and they are responsible to the in ltration of macrophages within the brain. A er activation of macrophages, it releases pro-in ammatory cytokines, which further promote the neurotoxicity and apoptosis in brain cells [19].

Microglial activation is inducted by pro-in ammatory molecules and related signaling pathways that leading to Aβ aggregation, tau formation, synaptic damage, neuronal loss and the activation of other in ammatory participants. Pro-in ammatory cytokines may have multiple roles both neurodegeneration and neuroprotection. It is di cult to know the precise role of pro-in ammatory cytokines in AD [20].

Immunotherapeutic Approach

Various immunotherapeutic approaches for AD are under research process. Synthetic Aβ42 evaluated in transgenic mice models with direct immunization and this evaluation has been provided the Aβ immunotherapy that stimulates the T-cells, B-cells and microglial immune responses. With active immunization administrated of synthetic fragment of Aβ conjugated to a carrier protein that responds the T-cell directly against antibodies. Last approach is passive administration with monoclonal antibodies directed against Aβ which under investigation. Other mechanisms like as plaque breakdown, peripheral sink and aggregation sink are trying as new immunotherapy of AD [21]. Active immunizations developed with minimize T-cell reaction and maximize antibody production but passive immunizations are being devised [22]. Various therapeutic approaches and their results are mentioned brie y in Table 1.

Many studies have shown that broad amyloid and tau pathologies

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